FORUM 2002

FOCUSSED OVERVIEWS, REVIEWS
AND UPDATES IN MEDICINE

SUNDARAM MEDICAL FOUNDATION
Dr. RANGARAJAN MEMORIAL HOSPITAL

IMAGE AUDITORIUM, Chennai

1,2,3 March 2002

CONFERENCE PROCEEDINGS

"A compilation of current views on topics of practical relevance"

Sundaram Medical Foundation
Dr. Rangarajan Memorial Hospital
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DEDICATION

FORUM 2002 is dedicated with deepest feelings of love and affection
to the guiding hand and sustaining spirit of

Dr S Rangarajan, MD
Founder Chairman & Managing Trustee
Sundaram Medical Foundation
Chennai

as one more step towards realization of his vision and dream
of developing his institution as a model of clinical
and academic excellence in this country.

FORUM 2002

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FORUM 2002

Friday 1 March 2002

WORKSHOP I: **COMMON OFFICE PROCEDURES FOR THE GP**

Objectives:	To equip the General Practitioner with knowledge and skills for carrying out common bedside procedures in the setting of the outpatient clinic
Content:	Lectures on equipment and infrastructure needed, multimedia demonstration of procedures, video presentations and hands-on workstations

Lectures include
Setting up a procedure area in a busy OPD
Asepsis & sterility - practical considerations
Tools of the trade – buying, maintaining & storing
Blood pressure measurement – advanced users guide

Multimedia & video demonstrations
Nasogastric insertion
Urethral catheterization
Rectal exam, proctoscopy, administration of an enema
Pleural & peritoneal tap
Venous access
Local anesthesia techniques
Pap smear
Diabetic foot exam

Hands-on session
Suturing, excision biopsy, I & D
Dressings, splints and plaster of Paris application
Urethral catheterization
Wound care

Faculty	Dept. of General Surgery, Internal Medicine, Orthopedics, Plastic Surgery and Urology of Sundaram Medical Foundation
Location	Hotel Radha Park Inn, Chennai.

FORUM 2002

Friday 1 March 2002

WORKSHOP II: **PEDIATRIC ACUTE THERAPY WORKSHOP**

Objectives: To equip General Practitioners with the skills needed for handling common acute pediatric conditions

Content: Consists of lectures, hands-on workstations and small group sessions. There will be plenty of time for one on one discussion where common doubts can be clarified

Lectures include
The child with stridor
The wheezing child
Stabilization and transportation of the acutely ill child
Scorpion stings and snakebite

Workstations include
Airway adjuncts – oxygen therapy, nebulization, pulse oximetry, Ambu bagging and cervical spine protection
Venous access and shock management, including intraosseous fluid administration
Common bedside procedures – NG tubes, Foley catheterization, i.v. catheters, enemas

Small group sessions include
Neurology – seizures and approach to altered mental status
Pediatric surgery – foreign bodies and the acute abdomen
Common neonatal emergencies

Faculty: Dept. of Pediatrics, Pediatric Critical Care, Critical Care and Anesthesia of Sundaram Medical Foundation and other prominent national and local pediatric sub specialists

Location: Hotel Radha Park Inn, Chennai.

FORUM 2002

Saturday 2, March 2002

8.50 am	**WELCOME**	
PEDIATRICS		
09.00 - 09.20 am	Viral hemorrhagic fever	Dr. Sarala Rajajee
09.25 - 09.45 am	The child with an incessant cry	Dr. S. Mahadevan
09.50 - 10.10 am	Lymphadenopathy in children	Dr. Y.K. Amdekar
10.15 - 10.30 am	Recognizing the acutely ill child	Dr. Bala Ramachandran
10.30 - 10.45 am	*AUDIENCE INTERACTION*	
10.45 – 11.15 am	TEA	
LABORATORY MEDICINE		
11.15 – 11.30 am	Urine examination	Dr. S. Suresh
11.35 - 11.50 am	Interpreting abnormal LFTs	Dr. Malathi Sathiyasekaran
11.55 - 12.10 am	*AUDIENCE INTERACTION*	
Dr.RANGARAJAN MEMORIAL ORATION		
12.15 – 01.15 pm	Towards a Family & Community Oriented GP-The elusive goal of medical education in India	Dr.Ravi Narayan
01.15 - 02.00 pm	LUNCH	
CARDIOLOGY		
02.00 - 02.20 pm	Cardiac failure	Dr. K. P. Misra
02.25 – 02.45 pm	Acute coronary syndrome	Dr. V. K. Menon
02.50 - 03.10 pm	Interventional cardiology	Dr. Sriram Rajagopal
03.15 - 03.30 pm	*AUDIENCE INTERACTION*	
INFECTIOUS DISEASES		
03.30 - 03.45 pm	Newer antibiotics	Dr. V.Ramasubramanian
03.50 - 04.10 pm	Case discussion : Chest Infiltrates Infection in inpatients	Dr. Ram Gopalakrishnan
04.15 - 04.30 pm	*AUDIENCE INTERACTION*	
NOT THE END OF THE ROAD		
04.30 - 04.45 pm	Ray of hope in dermatology	Dr. K. N. Sarveswari
04.50 - 05.15 pm	21st century dentistry	Dr. Vijaya Bharathi Rangarajan
05.20 - 05.35 pm	Cerebral palsy - one step at a time	Dr. Sudhakar Williams
05.35 – 05.50 pm	*AUDIENCE INTERACTION*	
05.50	TEA	

FORUM 2002

Sunday 3, March 2002

FLUID THERAPY

09.00 - 09.10 am	Capsule: intake & output chart	
09.15 - 09.30 am	Children	Dr. M. Vijayakumar
09.35 - 09.55 am	Perioperative patients	Dr. K. Sriram
10.00 - 10.15 am	Trauma and the critically ill	Dr. R. Rajaram
10.15 - 10.30 am	*AUDIENCE INTERACTION*	

PRACTICE GUIDELINES I

10.30 - 10.50 am	Asthma	Dr. S. Narasimhan
10.55 - 11.10 am	Adenotonsillitis: when do we operate?	Dr. K. KrishnaKumar
11.10 - 11.20 am	*AUDIENCE INTERACTION*	
11.20 – 11.50 am	TEA	

DIABETES

11.50 - 12.10 pm	Newer oral hypoglycemic agents	Dr. Radha Reddy
12.15 - 12.35 pm	Diabetes care "More than glucose"	Dr. Ambrish Mittal
12.40 - 01.00 pm	Case discussion : Impaired glucose tolerance test Pre-operative diabetic care	Dr. M. Satyajit
01.00 - 01.15 pm	*AUDIENCE INTERACTION*	
01.15 - 02.00 pm	LUNCH	

PRACTICE GUIDELINES II

02.00 - 02.15 pm	Dyslipidemia	Dr. Usha Sriram
02.20 - 02.40 pm	Osteoporosis	Dr. Ambrish Mittal
02.40 - 02.55 pm	*AUDIENCE INTERACTION*	

WOMEN'S HEALTH

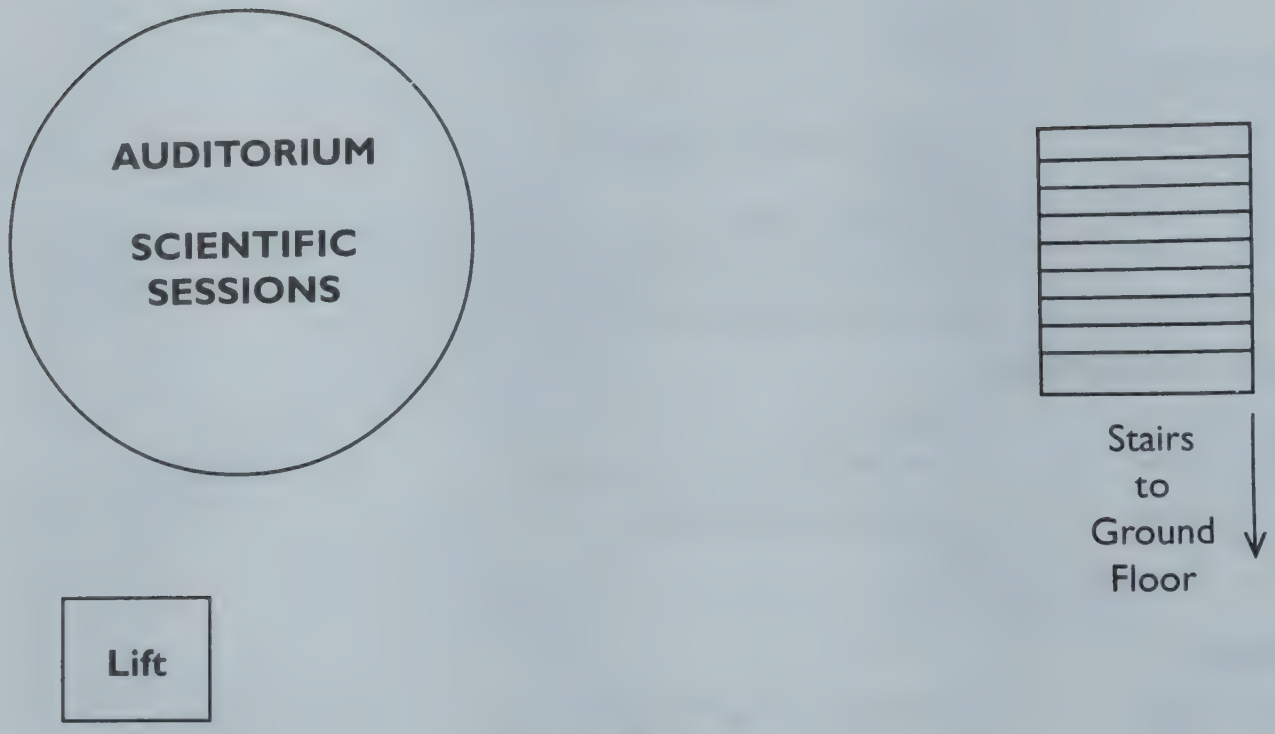
03.00 – 03.15 pm	Panel discussion: Pelvic pain Dr.V.P.Paily, Dr.V.K.Shantha,	Dr.G.S.Kailaash
03.20 – 03.35 pm	Practice guidelines: Pelvic pain	Dr.V.P.Paily
03.40 – 03.55 pm	Practice guidelines: Infections in pregnancy	Dr.Gita Arjun
03.55 – 04.10 pm	*AUDIENCE INTERACTION*	

ALTERNATIVE MEDICINE

04.10 - 04.40 pm	Co-ordinator: Dr. Arjun Rajagopalan Panelists: Dr. A.U. Ramakrishnan and Dr. Krishna Raman
04.40 - 05.10 pm	**QUIZ**
05.15 - 05.30 pm	**VALEDICTORY FUNCTION**
05. 30	TEA

FORUM 2002

VENUE MAP



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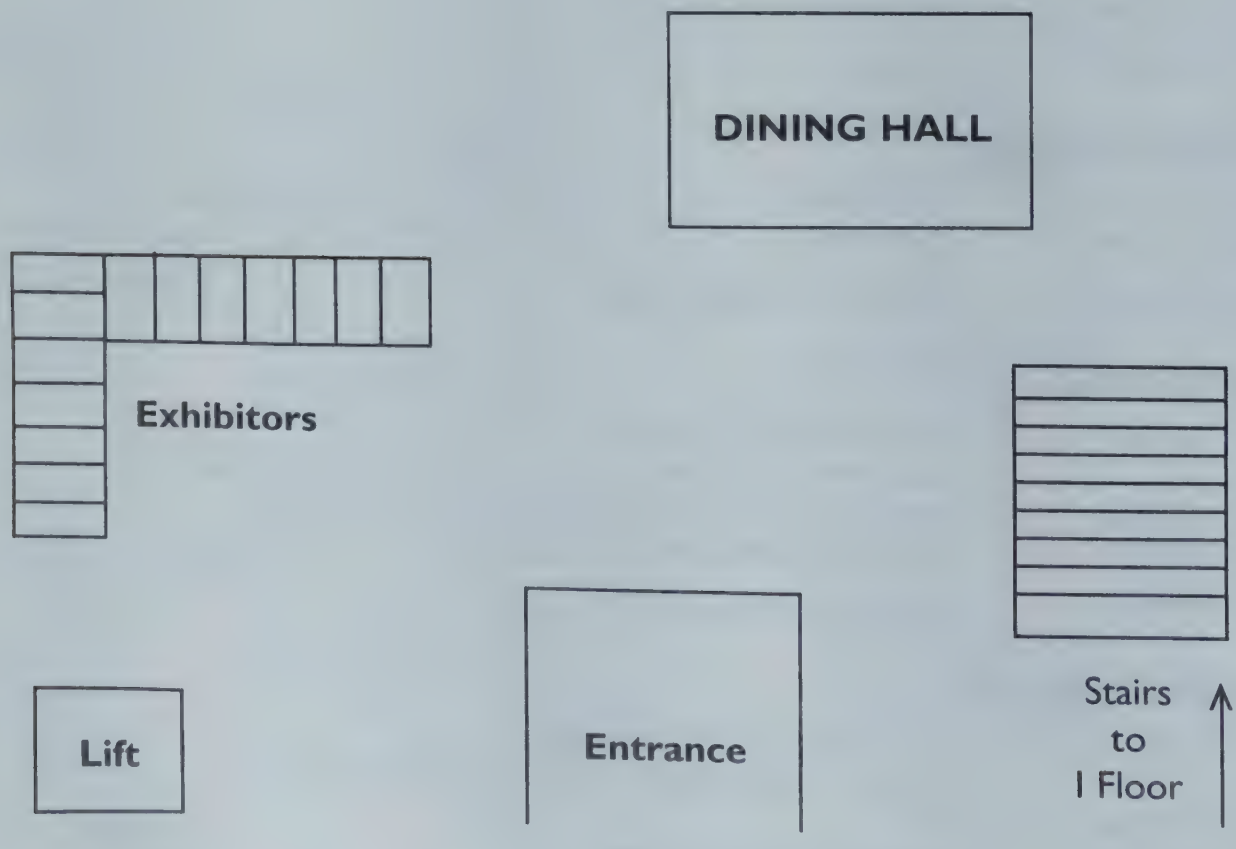

GROUND FLOOR

FORUM 2002

MARCH 2-3, 2002

From the desk of the Organising Secretary

Dear Friend,

At the outset, I welcome you to FORUM 2002, Focused Overviews, Reviews and Updates in Medicine, the conference designed for the family physician. Most of you who have attended FORUM '98,'99,2000 & 2001 have returned for FORUM 2002. We are aware that FORUM owes its success to all of you. I extend a special welcome to you. FORUM 2002 is designed for all doctors in our professional milieu since they are often faced with problems outside their chosen speciality.

We have responded to the requests from the delegates who attended the previous conferences and have included topics on Laboratory Medicine, Cardiology, Infectious diseases, Fluid Therapy, Practice guidelines, Women's Health & Alternative Medicine relevant to the General Practitioner. A panel of experts will handle common problems in Paediatrics & Gynaecology. Popular segments such as the Interactive case discussion and the Quiz have been retained. The Dr.Rangarajan Memorial Oration this year will be on "Towards a Family & Community oriented GP - The elusive goal of medical education in India".

There is a Pre-conference workshop on Common Office Procedures for the GP and Pediatric Acute Therapy. All topics have been chosen with care to represent the essence of Primary care, an area close to the heart of our dear departed Founder Chairman & Managing Trustee Dr. S. Rangarajan.

Like FORUM '98,'99, 2000 & 2001 this venture too has had the whole hearted blessings and encouragement - moral, financial, material and otherwise - of our Managing Trustee Mr. R. Ramanujam, our Trustee and Chief Executive Mr. S. Ravindran, the Chief of Medical Staff Dr. Arjun Rajagopalan and Executive Director Dr. Mrs.Vijaya Bharathi Rangarajan. I am extremely grateful to all of them, to our group companies & to all our sponsors from different segments of the health care industry for their support. I am also grateful to the effort of each and every one of my colleagues and other staff members at SMF, particularly to Dr. S. Suresh, the Joint Organising Secretary.

I also wish to thank the "SMF Family" and the conference secretary Miss. R. Anuradha for their untiring work.

The proceedings of this conference are being handed over to you in print. We hope you find this conference and the workshops educative and informative. Looking forward to see all of you at FORUM 2003.

Dr. S. Ramakrishnan
Organising Secretary

DENGUE HAEMORRHAGIC FEVER

Dr. Sarala Rajajee MD, DCH, DNB, PhD.
Senior Consultant – Paediatrics and Haematology
Kanchi Kamakoti Childs Trust Hospital
Chennai

Clinical profile and management

Dengue fever, a disease prevalent in South East Asian countries, Cuba, Venezuela and parts of India mostly among healthy children, is an acute febrile illness caused by four serotypes of Dengue virus and characterised by biphasic fever, myalgia or arthralgia, rashes, leucopenia and lymphadenopathy. Dengue haemorrhagic fever is characterised by haemoconcentration, by abnormalities of hemostasis and in severe cases, by a fluid and protein losing shock syndrome (Dengue shock syndrome – DSS). The viruses are arthropoid borne and daytime biting mosquitoes are the principal vectors, breeding in water stored for drinking and bathing. Female mosquitoes have greater potency than males due to repeated blood meals. There is no cross protection between the 4 Dengue serotypes, but there is cross- reaction.

Pathophysiology of DHF/DSS

The incubation period of DHF/DSS is unknown but is presumed to be that of dengue fever - 2 to 7 days. There is increased vascular permeability with abnormal hemostasis.

Previous immunologic experience

The Central risk factor in DHF/DSS is the circulation of pre-infection antibody whether from prior dengue (? or other Flavivirus) infection or passively acquired from the mother. DHF/DSS does not occur in all secondary dengue infections. This is perhaps due to a requirement for specific sequences of dengue virus infections to produce shock. It has been reported in various studies that nearly all DSS cases occur with secondary dengue infection. DSS rarely occurs with primary dengue infections. Dengue 2 strains have been reported to possess unusual virulence which were expressed in the presence of antibody.

Epidemiologic, clinical and virologic studies have shown a significant association between severe illness and infection in the presence of circulating dengue antibody whether acquired passively from the mother or from a previous infection.

The circulating antibody has two biologic activities, viral neutralisation and infection enhancement. DHF/DSS occurs when the neutralising antibody has catabolised to a low titre leaving only infection enhancing antibodies in circulation. Dengue virus multiplies in culture of human mononuclear phagocytes that have very small quantities of dengue antibodies. It has been suggested that the number of infected mononuclear cells in individuals with naturally or passively acquired antibody may exceed that in

nonimmunes. Therefore increased production of infected cells may contribute to shock, possibly through the release of cytokines.

Early in the acute phase of secondary dengue infection, there is rapid activation of complement system either alternate or classical pathway. During shock, blood levels of C1q, C3, C4, C5-8, C3 proactivators are depressed and C3 catabolic rates increased. The blood clotting and fibrinolytic systems are activated through the activation of complement. Shock may be mediated by the peptides C3a and C5a. Capillary damage allows fluid, electrolytes, proteins and in some instances red cells to leak into the extravascular spaces, leading to hypovolemia, haemoconcentration, increased cardiac work, tissue hypoxia, metabolic acidosis and hyponatremia.

Platelets

The second consistent attribute of DHF is thrombocytopenia. Bone marrow taken on day 4 after the onset of fever will be found to be hypocellular with reduced megakaryocytes. Later there is progressive return to normal cellularity.The mechanism of increased destruction of platelets during dengue infection is not known. Active complement components, dengue virus itself, damaged endothelial cells and activation of blood clotting system remain as possible mechanism of thrombocytopenia.

Coagulation factors

DHF is characterised by prolonged bleeding time, prolonged prothrombin time, activated partial thromboplastin time, decreased fibrinogen levels and increased fibrinogen degradation products. Prolonged PTT is reported in 54% of cases. Prolonged PT in 33% and normal thrombin time in most cases of DHF.

Pathology

No gross or microscopic lesions have been found that might account for deaths. In rare instances, death may be due to gastrointestinal or intracranial bleeds. 97% shows evidence of cerebral oedema, 76% had pleural effusion and 74% had ascites. Livers are enlarged with fatty degeneration, focal necrosis, hyaline necrosis of Kupffer cells and subcapsular haemorrhages. The heart shows subendocardial haemorrhages. Bone marrow shows depression of all elements. There is immune complex glomerulonephritis, which clears in 3 weeks.

Clinical manifestation of dengue

Infants and children infected with dengue virus for the first time (primary dengue infection) develop simple fever indistinguishable from other viral infection. Maculopapular rash may accompany the fever or may appear during defervesence. Dengue fever is an acute biphasic fever with headache, myalgia, arthralgia, rash and leucopenia.

DENGUE HAEMORRHAGIC FEVER

Case definition includes fever, hemorrhagic manifestations, thrombocytopenia (100,000/m3 or less) haemoconcentration (haematocrit 20% of recovery value) or objective evidence of increased capillary permeability (serous effusion), hepatomegaly.

DENGUE SHOCK SYNDROME includes all above criteria plus hypotension or narrow pulse pressure (20mm hg or less)

Gradation of DHF severity

Grade I: Fever, non-specific symptoms, and positive tourniquet test
Grade II: Spontaneous bleeding skin and mucous membranes
Grade III: Circulatory failure, rapid weak pulse, narrow pulse pressure, hypotension, cold clammy skin, restlessness
Grade IV: Profound shock, undetectable BP and pulse

CNS manifestation of DHF/DSS

They include fits, spasticity, altered sensorium or behaviour more than 8 hours after correction of shock, transient paresis and encephalitic picture. CSF and ultrasound head are normal. These features are usually due to cerebral oedema and are reversible with proper treatment

Laboratory findings

Normal WBC or leucopenia is common initially with neutrophils predominating towards the end of febrile phase; a relative lymphocytosis with more than 15% atypical lymphocytes is common (15 – 50%). Thrombocytopenia and haemoconcentration are constant findings. Clotting abnormalities are common and appear to correlate with disease severity.

Hypoproteinemia particularly hypoalbuminemia is common. There is hyponatremia in severe cases. Serum levels of SGOT, SGPT are often elevated. Evidence of plasma leakage is seen as pleural effusions (right more than left). ECG abnormalities have been reported in 44% of children with DHF. This may be secondary to myocardial involvement, which in turn is related to severity of the disease. The most common findings are ST – T wave changes or sinus bradycardia.

Management – general consideration

The major pathophysiological abnormality seen in DHF/DSS is an acute increase in vascular permeability leading to leakage of plasma and later to hypovolemic shock if loss of plasma is critical. Hypovolemic shock leads to tissue anoxia, metabolic acidosis and death if uncorrected. With volume

replacement there is dramatic recovery indicating that there is no destruction or inflammatory vascular lesions. Short acting pharmacological mediators (C3a, C5a) and cleavage products of complement play an active role in the pathogenesis of increased vascular permeabilty.

Treatment

The most important therapy is volume replacement. In dengue fever this can be done with oral electrolyte solution or fruit juices. The patients with cool periphery, acute abdominal pain or oliguria need parental fluids. The volume and the type of fluid is similar to that used in diarrhoea with moderate isotonic dehydration. But the rate should be carefully titrated.

The required fluid volume should be charted on a 2 to 3 hourly basis and rate of administration adjusted throughout the 24 to 48 hour period of leakage. Frequent recording of vital signs is needed for adjusting fluid replacement. Excessive replacement will cause respiratory distress (from massive pleural effusion and ascites), pulmonary congestion and oedema.

Type of fluids

Crystalloids	Ringer lactate
Colloids	Platelet rich plasma
	Fresh frozen plasma
	Fresh blood

Immediate replacement at 10 to 20 ml/kg/hr or in profound shock as bolus of 10ml/kg 2 to 3 times. In case of continued shock, colloid may be given at 10ml to 20ml/kg. Fresh blood may be given as it replaces plasma volume and increases the intravascular oncotic pressure as well as replaces platelets and coagulation factors. Frusemide may be combined with blood transfusion to avoid fluid overload due to transfer of fluid from extravascular to intravascular compartment due to increased intravascular oncotic pressure.

Continued replacement of further plasma loss

Intravenous fluids are continued with the rate adjusted to the rate of plasma loss as guided by vital signs for a period of 24 to 48 hours. In severe cases of shock, central venous pressure will have to be monitored. Reabsorption of extravasated plasma takes place 1 – 2 days thereafter (manifested by clearing of pleural effusion and ascites) and may cause hypovolemia, heart failure and pulmonary oedema if more fluid is given.

Correction of electrolyte and metabolic disturbances

Hyponatremia and metabolic acidosis occur in severe cases. Electrolyte and blood gases should be determined periodically in severely ill patients and those with refractory shock.

Management of patients with unusual manifestations

CNS complications

This infant may present with repeated convulsions associated with features of DHF/DSS. After initial correction of shock with volume replacement and anticonvulsants, anticerebral oedema measures such as intravenous frusemide is needed

Renal failure

There are occasional patients with renal failure after prolonged shock, which need appropriate treatment. Dialysis is rarely needed. Steroids were not found to be useful in the management. With careful monitoring of patients and appropriate volume replacement as described the mortality can be brought down considerably.

The annual epidemic starts from October to February each year. Apart from features of DHF/DSS, neurological manifestations have been a prominent feature. Viral studies have shown Dengue 2 to be the pathogen in these children. A high index of suspicion and timely therapeutic intervention in these children is needed to avoid case fatality.

THE CHILD WITH AN INCESSANT CRY

Dr.S.Mahadevan MD,PhD,MNAMS.

Professor of Paediatrics

JIPMER, Pondicherry

Crying in a child is associated with any condition causing pain or discomfort. In some the illness is apparent and in many it is difficult to explain. Acute disorders such as otitis media, intestinal cramping with diarrhea, corneal abrasion, a hair tourniquet on a digit, and incarcerated hernia are usually ruled out by history, as is the chronic condition of GE reflux

Immediate evaluation should focus on infection (sepsis, meningitis), intra-abdominal conditions (bowel obstruction, appendicitis,) metabolic disturbances (hypernatremia, Vitamin A poisoning) cardiac dysrhythmias, seizures, respiratory failure and increased intracranial pressure. Sepsis should be considered even in the absence of fever. Repeated evaluations of the infant are necessary in this situation to ensure that abdominal, cardiac, respiratory and neurological status is not changing. The evaluation requires a careful and thorough approach. A cause that is treatable or empiric treatment of life-threatening illness must be the focus of our evaluation.

Primary incessant or excessive crying in the absence of an illness is a diagnosis of exclusion. Incessant crying is defined in a young child otherwise healthy and well fed as paroxysms of fussing, or irritability or crying lasting for a total of more than three hours a day and occurring on more than three days in any one week. This is due to a poor misfit between a sensitive child and poor parental handling technique. Soothing the infant with repeated sound, stimulating less by decreased picking up and feeding in response to every cry are useful. A dimly lit environment, with minimal handling and correction of faulty feeding technique without change in the composition of feeds are found beneficial. Drugs are unnecessary. Antispasmodics and simethicone, though popular, are not of benefit. Medication should be optional. In extreme cases with sleep deprivation in parents, diphenhydramine 5 mg twice daily for one week to the child is sufficient. If excessive crying recurs it may be repeated for one more week. As a last resort, stay of the child with a competent relative away from the parents has reduced crying sufficiently in many. Parents must never be made to feel inadequate and they need to be assured that crying will diminish considerably in 2-3 days. Counseling is the most cost effective approach to help parents to cope with such infants.

LYMPHADENOPATHY IN CHILDREN

Dr.Y.K.Amdekar MD,DCH.
Hony Professor of Paediatrics
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Lymph nodes commonly enlarge in response to infection in the draining area or generalized systemic infection and less often due to infiltrative disorders. Minor recurrent infections around head, neck and face result in bilateral, small, discrete glands in the posterior cervical region and are considered insignificant. Similar glands in the inguinal regions are also commonly found in children. However, if lymph nodes are > 1 cm in size or are increasing in size over time, they are considered to be pathological. Besides, other features such as abnormal consistency, tenderness, matting or lymph nodes in an unusual area suggest pathology. Lymphadenopathy < 6 months of age is rare except BCG lymphadenitis and is invariably pathological.

Common causes of lymphadenopathy include acute and chronic infections and lymphoreticular malignancy. Acute infection may be bacterial or viral. Acute bacterial lymphadenitis presents with fever and tenderness localized to a single area and may form an abscess. Acute viral infection generally leads to multiple lymph nodes with or without hepatosplenomegaly as in case of infectious mononucleosis or rubella respectively. Acute lymphatic leukemia may present with generalized lymphadenopathy of acute onset and is accompanied with hepatosplenomegaly, pallor, and bleeding and bony tenderness. Chronic infection may present as a single enlarged lymph node as in tuberculosis. Lymphoma may also present similarly. Chronic disseminated lymphadenopathy is a feature of generalized tuberculosis or HIV infection, in which hepatosplenomegaly may also be present.

Clinical Approach

Onset, progression and accompanying symptoms such as fever, pain, skin rash and loss of weight help to consider the etiology. Physical examination defines sites of involvement, tenderness, hepatosplenomegaly, pallor, and bony tenderness. Investigations may include haemogram, tuberculin test, chest x-ray, abdominal ultrasound and finally lymph node biopsy. Specific serological tests may be necessary to diagnose infection. Bone marrow may be necessary in case of suspected leukemia.

Treatment

Benign laryngomalacia recovers over time usually within 6-9 months and needs no treatment. Rarely it is so severe that child may not be able to maintain gas exchange well. Only in such situations, it

may require surgical treatment. Bacterial infections can be treated with appropriate antibiotics. Viral infections are usually self-limiting with the exception of HIV infection, for which anti-retroviral drugs are recommended. Leukemia and lymphoma are treated with specific chemotherapy. BCG lymphadenitis is relatively benign condition and may be left alone, unless the gland continues to enlarge and soften; in which case, surgical excision may be all that is necessary. Anti-TB drugs may not be necessary.

Algorithm:

Acute			**Chronic**	
Single	Multiple		Single	Multiple
Painful Bacterial	Painful Viral	Painless Leukemia	Tuberculosis BCG Lymphoma	Tuberculosis Viral

RECOGNIZING THE ACUTELY ILL CHILD

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Unlike adults, cardiopulmonary arrest in children is rarely a sudden event. It is often the end result of progressive deterioration in respiratory and circulatory function. Irrespective of the initiating event, the final common pathway is the development of cardiopulmonary failure and possible cardiopulmonary arrest. Survival following respiratory arrest ranges from 75% to 90% - however, once asystolic cardiac arrest ensues the survival is only 7 – 11%. Therefore, it is important to recognize a child who is in impending cardiopulmonary failure and intervene before the development of asystole.

Irrespective of the primary complaint, every examination should consist of a systematic evaluation of the **Airway**, **Breathing** and **Circulation.** With experience, it is possible to recognize potential respiratory failure and shock within 30 seconds. It is not necessary to rely on laboratory tests or X-Rays.

Airway

Every child with clinical signs of respiratory distress has *Potential Respiratory Failure*. If the child does not improve with therapy, such as positioning, oxygen and suctioning, he has *Probable Respiratory Failure*. Based on a quick examination, the Airway should be classified as:

- Clear (no assistance needed)
- Maintainable (assistance such as positioning, suctioning or bag-mask ventilation needed)
- Unmaintainable (needs invasive support, such as intubation)

Breathing

- **Rate:** Tachypnea is usually the first sign of respiratory distress. Though respiratory rates differ with age, a rate of more than 60 per minute is abnormal at any age. Also, a slow or irregular rate in an acutely ill child is dangerous
- **Effort:** This assesses the work of breathing – look for the presence of chest retractions, nasal flaring and use of accessory muscles of respiration. Head bobbing in an infant often indicates that the child is about to tire out.
- **Air Entry:** Evaluate the chest expansion visually and auscultate the breath sounds
- **Skin Color:** Look for cyanosis or a gray or ashen color, all of which can indicate poor oxygenation

Circulation

The Heart Rate, Systemic Perfusion and Blood Pressure should be evaluated

Heart Rate

Normal heart rates vary with age and an increased heart rate is a nonspecific sign of cardiovascular compromise. The heart rate should be evaluated in conjunction with the clinical condition, since fever, anxiety or pain can also increase the rate.

Systemic Perfusion

- **Pulses:** Weak or absent pulses are often present in shock. Hypotension often develops before loss of central pulses
- **Skin Perfusion:** Cool extremities suggest inadequate cardiac output. Normal capillary refill tie should be less than 2 seconds (in a warm environment). Pale, blue or mottled skin may also indicate poor perfusion
- **Mental status:** An altered mental status may indicate decreased cerebral oxygenation or perfusion. The level of consciousness can be rapidly assessed by the **AVPU** technique – **A**: Awake; **V**: responds to voice; **P**: responds to pain; U: unresponsive. A child who does not recognize the parents is often seriously ill.
- **Urine output:** Normal urine output is 1 –2 ml/kg/hr. This is an excellent indicator of renal perfusion and an hourly measurement with a catheter is more useful than a one-time assessment.

Blood Pressure

The lower limits of systolic blood pressure are:

0 – 1 month	60 mm Hg
1 month – 1 year	70 mm Hg
> 1 year	70 + (2 x age in years) mm Hg

A normal blood pressure does not automatically mean that all is well – it simply indicates that the child may be in a state of compensated shock, whereas a low blood pressure indicates that the shock is decompensated.

Based on this quick assessment, it is possible to determine whether a child is Respiratory Failure or Shock. One can then decide on the initial priorities of management depending on the physiological status of the patient.

URINE EXAMINATION

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Urine examination is a simple, inexpensive investigation that provides valuable clues in the diagnosis and management of renal and urinary diseases – of both intrinsic and systemic etiology. It includes

a. Urine analysis by dipstick and microscopic analysis
b. 24 hour urine tests

Urine analysis

Specimen collection is ideally from a midstream specimen with minimal contamination. High osmolality and low Urine pH preserves cellular elements in urine. These characteristics, which are present in the morning urine favors detection of glomerular disease. About 10 ml of urine is required and is tested by dipstick analysis and microscopic examination.

Appearance

Color change – It may be due to hematuria or uncommonly due to hereditary metabolic diseases. Do not overlook drug history & food colorings e.g., Pyridium, Rifampicin, Riboflavin, Rhubarb

a. Tests using dipstick

Specific gravity

Normal 1.010 to 1.030. It is used as a substitute for osmolality. Presence of protein or glucose may affect specific gravity without affecting osmolality.

Glucose

It is commonly practiced to diagnose and monitoring control of diabetes mellitus. But serum monitoring of glucose is relatively better.

Protein

The concentration of urine determines strength of reaction. This is probably most impactful information that should be used to determine target organ damage in patients with hypertension and diabetics who are at risk of renal disease. Proteinuria has prognostic significance and therapeutic relevance

in systemic diseases with proteinuria and glomerular. Stress, fever and infections may cause transient proteinuria Dipstick tests may not detect protein other than albumin.

Microscopic

Urine would need to spun prior to examination – at 2500 rpm for 5 minutes prior to examination. A simple viewing microscope with × 100 & × 400 times magnification would be adequate to use for diagnostic purposes. Epithelial, tubular, white blood and red blood cells may be seen under microscope. Familiarity with cellular morphology is helpful to identify the types of cells. Microscopic examination is essential to detect renal disease for patients with edema and hypertension. In addition it is useful to assess response to treatment

Cells

RBCs

Small round nonnucleatd cells. Normal: 1-2/hpf. Increased in Glomerulonephritis, Urinary tract infection. Usually not seen in diabetic nephropathy

WBCs

Slightly larger than rbcs. Normal 2-4/hpf. Increase in urinary tract infection, glomerular diseases, interstitial nephritis

Epithelial cells

Largest of the cells in urine. Indicates that urine collection was inappropriate and contaminated by urethral elements

Casts

It consists of matrix of Tamm- Horsfall protein - a urinary mucoprotein mixed with cells

Hyaline casts: Consist of mucoprotein only

Granular casts: Finely and granular material derived from altered serum proteins. Occurs in Acute **Tubular Necrosis:** Tubulointerstitial nephritis & Glomerulonephritis

Rbc casts: It indicates renal parenchymal bleeding. Occurs in glomerular diseases. Color change may occur over time so that rbc casts may resemble granular casts.

Wbc casts: Occurs in pyelonephritis and tubulointerstitial nephritis.

b. Tests using Multistix

Additional information may be obtained by using urine dipstick with reagents to detect other chemical substances of pathological and clinical importance

Ketone: Dipsticks detect acetoacetate only. Levodopa may cause false positive reaction.

Bilirubin: False positive – Chlorpromazine. False negative – Vitamin C

Urobilinogen: Normally present. Absence indicates presence of obstructive jaundice

c. Microalbumin

This indicates small amounts of protein excretion that may not be detected by standard dipstick. It is used to detect the clinical phase that precedes onset of proteinuria. Cost factor is a major issue limiting wide application.

d. Spot Urine analysis

Used to quantitate proteinuria. Normal protein excretion upto 150mg/24hr. Spot protein and creatinine is a substitute for 24 hour analysis. Protein and creatinine levels are reported as mg%. The ratio represents the number of grams of protein excreted in 24 hours

24-hour urine analysis

Proteinuria

24-hour urine analysis is mainly used for protein estimation. Creatinine estimation may be added to assess whether urine collection is complete. Normally in 24 hours 15-20 mg/kg of creatinine is excreted in urine in 24 hours. Relevant in diagnosis of glomerular diseases, diabetics and hypertensives. Assessment is useful to detect target organ damage and also therapeutic response.

Stone disease

24-hour urine Calcium, uric acid and phosphorus are done to study excessive excretion of potential stone forming substances

Calcium – 4mg/Kg

Uric acid – .7 to 1 mg/Kg

INTERPRETING ABNORMAL LIVER FUNCTION TESTS

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The liver performs a variety of important biochemical, synthetic and excretory functions. Laboratory tests often referred to as "Liver function tests" LFTs is a misnomer as the majority of these tests assess liver cell damage rather than function. These tests lack sensitivity, specificity and prognostic power. To increase both the sensitivity and specificity, it is essential to use them as a "battery" rather than "singly"

The tests are grouped as those that:-

I Reflect hepatobiliary injury

II Measure the capacity of the liver to transport organic anions

III Measure the capacity of the liver to metabolize drugs.

IV Measure hepatic synthetic function

V Detect chronic inflammation or altered immune response

VI Miscellaneous

I a) Tests that reflect injury to hepatocytes

The **aminotransferases aspartate aminotransferase** (AST) or SGOT and **alanine aminotransferase** (ALT) or SGPT are relatively good indicators of liver cell membrane damage. ALT is primarily from liver cytosol, whereas AST is distributed in a wide variety of tissues - liver (cytosol & mitochondria), muscle, kidneys, brain, pancreas, lung, etc., The normal range is 30-40 IU/L.

Elevated transaminases

a) Marked: (>20 fold) in circulatory shock, toxic and viral hepatitis.

b) Moderate: (3 to 20) in chronic hepatitis, alcoholic hepatitis, acute biliary obstruction.

c) Mild: (2-3 fold) NASH myositis, hepatic neoplasm.

Decreased transaminases

a) Long-term dialysis.

Falling levels

a) Recovering patient

b) Rapid fall may indicate poor prognosis in patients with FHF

Fluctuating levels

Chronic hepatitis especially type C.

Ratio of AST/ALT

a) If more than 2 indicate ethanol injury.

b) In viral hepatitis, the ratio is <1.

The limitations of transaminases are

a) 6% of healthy a symptomatic people have abnormal liver enzymes.

b) Non hepatic cause increase transaminases (Myocardial infarction, myositis)

c) Aetiology not possible

d) Does not correlate which severity

e) Cannot predict outcome

The transaminases are therefore used in screening for liver cell injury and monitoring therapy in chronic hepatitis.

(ii) **Lactate Dehydrogenase**:- This enzyme has wide distribution and elevations are seen in acute and chronic liver disease, skeletal and myocardial injury, renal infarction.

I b) Tests that indicate cholestasis

i) **Alkaline phosphatase**. This family of immunologically distinct isoenzymes is distributed in abundance in placenta, ileal mucosa, kidney, bone & liver. In the liver it is present in the canalicular membrane and bile duct epithelium. Cholestasis stimulates synthesis and bile salts facilitate release from cell membranes.

Increased levels:

a) Childhood, adolescence

b) Pregnancy

c) Fatty meal

d) Cholestatic disorders (extra hepatic, intra hepatic)

e) PBC, infiltrative liver disease.

In obstructive jaundice the levels are high for almost a week after relief of obstruction (half life – 7 days)

Low levels:

Wilson's disease which FHF & hemolysis

Hypothyroidism

Pernicious anemia

Zinc deficiency

Limitations of SAP

It does not differentiate between hepatic and non-hepatic sources, extra hepatic and intra hepatic obstruction.

ii) **Gamma Glutamyl Transpeptidase (γGTP)**: Localised in the entire hepatobiliary tree and levels parallel SAP. It is useful to detect surreptitious alcohol ingestion and along which SAP in cholestasis.

iii) **5' Nucleotidase:**-This enzyme is found in the liver in association with canalicular and sinusoidal plasma membrane. The increase in liver parallels SAP. It helps to differentiate physiological from pathological increase in SAP.

II Tests that measure capacity to transport organic anions.

i) **S. bilirubin**. The estimation of total and fractionation of bilirubin indicates the capacity of the hepatocyte to take up, conjugate and excrete bilirubin. In normal individuals the S. bilirubin is almost all unconjugated (0.1 to 1mg/dl). In hemolytic jaundice, CCF, Gilbert's syndrome the jaundice is predominantly unconjugated. Conjugated hyperbilirubinemia is seen in biliary obstruction. In Dubin Johnson and Rotor syndrome the direct bilirubin is increased without rise in SAP.

ii) **Urine bilirubin**: Only direct reacting conjugated bilirubin is detected in the urine. The test is useful in early detection of acute viral hepatitis. The absence of bile pigments in a patient which jaundice could indicate recovery or unconjugated hyperbilirubinemia.

iii) **Urine urobilinogen**: The levels are increased in hemolysis and slightly elevated in hepatocellular dysfunction. Urobilinogen is absent in extra hepatic bile duct obstruction.

iv) **Serum bile acids**: Though they are specific for liver disease in contrast to enzymes such as AST or SAP, its sensitivity over serum bilirubin is still controversial and is not yet used in routine practice.

III Tests that measure the capacity of the liver to metabolize drugs.

The clearance and breath tests such as a) Aminopyrine breath test, b) caffeine clearance, c) galactose clearance test are not used routinely,

d) **Lidocaine Metabolite Formation:** Lidocaine is metabolized to monoethylglycine xylidide (MEGX) within the hepatic cytochrome P450 system. Cirrhotics with higher MEGX fare better than those with lower MEGX levels.

IV Tests that measure hepatic synthetic function.

The liver synthesizes several proteins including albumin, coagulation factors and lipoprotein.

1) **Albumin:** An important plasma protein synthesized exclusively by the liver. Normal serum values range from 3.5 to 4.5gms/dl. Albumin has a long half-life of 20 days. The level depends on rate of synthesis, degradation and volume of distribution. Decreased albumin is seen in PEM, protein losing enteropathy, chronic infections, nephrotic syndrome. In liver disease decreased albumin < 3gms should raise the suspicion of CLD.

2) **Prothrombin Time**: A useful test to measure the synthesis of some of the coagulation factors I, II, V, VII & X. Normal 9-11secs. It is abnormal if prolonged 2secs more than control. If prothrombin time returns to normal or improves by at least 30% after a single dose of vitamin K it may be surmised that the parenchymal function is good.

3) **APTT**: Abnormal APTT indicates deficiency of factors VIII & IX. The test is more sensitive than Prothrombin Time.

4) **Lipoprotein**: Liver is the major source of plasma lipoprotein except chylomicrons. In cholestasis, cholestrol, phospholipids & lipoproteinX are increased.

V Tests that detect chronic inflammation or altered immune regulation

Serum immunoglobulins are produced by stimulated β lymophocyte and thus do not test liver function directly. Their elevation in many patients with CLD is due to impaired function of RE cells is hepatic sinusoids. In chronic liver disease there is a diffuse polyclonal increase in immunoglobulin. These laboratories tests suggest but seldom make a specific diagnosis. Further tests such as serology, ultra sound, liver biopsy and cholangiography may be required.

LIVER FUNCTION TESTS – INTERPRETATION IN VARIOUS DISORDERS

Examples	AST/ALT	Alk. Phosp.	Bili/fract	Prothrombin	Albumin	Globul
Toxic/ischemia	50-100x	1-3x	1-5x	Prolonged if>5secs and not corrected by vit K suggests poor prognosis	Normal	Norma
Viral (A or B)	5-50x	1-3x	1-30x	-do-	Normal	Norma
Alcohol	2-5x AST/ALT >2	1-10x	1-30x	Prolonged	Decreased	Increas γ globu
Cirrhosis	2-5x	1-3x	1-3x	Prolonged fails to correct with vit K	Decreased	-do-
Intrahepatic cholestasis	1-10x	1-4x	1-10x Both fractions elevated	Normal if prolonged will correct with vit K	Normal/ decreased	γ Globu norma
Complete biliary obstruction	1-5x	2-20x	1-30x	-do-	Normal – decreased in chronic disease	β-Globul may b increas
Partial biliary obstruction	1-5x	2-10x	1-5x	-do-	-do-	
Hepatic infiltration	1-3x	1-20x	1-5x	Normal	Normal	Usuall normal globul may b increas
Hemolysis **Gilberts**	Normal	Normal	1-5x, 85% indirect	Normal	Normal	Norma

CARDIAC FAILURE

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Cardiac failure is one of the commonest causes of death, not only in cardiac patients but also in other diseases. Almost all cardiac diseases at the end stage or due to severity and/or complications can lead to cardiac failure and decompensation of the heart.

Morbidity and mortality due to heart failure is very high and it depends on severity of the heart failure and the underlying disease. Not more than 20% of severe heart failure patients will survive beyond three years from the time of diagnosis.

Clinical diagnosis of heart failure is relatively easy by raised venous pressure, hepatomegaly, peripheral oedema and lung signs with crepitations and/or rhonchi. However subclinical or haemodynamic heart failure can be diagnosed by sophisticated investigations including echocardiogram and other special tests.

Over the years understanding and management of heart failure have changed dramatically. For example, diastolic heart failure was not even known about 20 years ago. It occurs in about 30 to 40% of cases in clinical practice. The following evolutionary changes have occurred in last four decades in the recognition and management of heart failure, which will be highlighted in detail in our presentation.

Cardiac Failure

1960-70's	2000's
1. Diastolic Failure unknown	1. Diastolic Failure well recognised
2. Digitalis and Diuretics only treatment	2. Many better and effective treatments
3. Beta blockers contraindicated	3. Beta blockers indicated
4. Ace Inhibitors not available	4. Ace Inhibitors available and strongly indicated
5. Vasoconstrictors like noradrenaline commonly used	5. Vasoconstrictors contraindicated. Vasodilators especially Inodilators used
6. Interventions like PTCA and CABG not available. Mortality approx. 80% in cardiogenic shock	6. Available and performed as life saving procedures. Mortality in cardiogenic shock is 20-30%
7. Prognosis is extremely poor.	7. Prognosis is much better but still unsatisfactory.

ACUTE CORONARY SYNDROME

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By definition chest pain is a symptom of an acute coronary event, now more accurately termed acute coronary syndrome (ACS). This syndrome includes unstable angina, non-ST segment myocardial infarction and ST-segment elevation myocardial infarction. All ACS episodes show the same underlying pathophysiological events: an imbalance in myocardial supply and demand. The imbalance has 5 potential causes:

- Non-occulusive thrombus on pre-existing plaque
- Dynamic obstruction such as vasospasm
- Progressive mechanical obstruction without evidence of underlying clot or vasospasm
- Inflammation or infection
- Secondary angina (anaemia, fever, thyrotoxicosis, hypoxemia)

By far the most common cause of ACS is thrombus formation. However the clot formation and its breakdown occur simultaneously. The dynamic nature of clot formation and degradation accounts for on-again off-again symptoms.

In the past, clinicians have difficulty diagnosing myocardial infarction without ST segment elevation or Q waves on the ECG. They'd rely on key cardiac markers such as CPK/CPK-MB & LDH to make to diagnosis. Waiting for these tests delayed treatment. The new guidelines encourage the physician to begin treatment immediately based on chest pain and ECG changes rather than cardiac markers. There is a high likelihood of ACS when any of the following exist;

- Chest and left arm pain/discomfort as the chief symptom with prior documented angina
- History of coronary artery disease
- Transient mitral regurgitation, hypotension, diaphoresis, pulmonary oedema and basal crackles
- New transient ST segment changes or T wave inversions with symptoms increased cardiac markers

Intermediate likelihood of ACS would be:

- Chest pain or discomfort
- Age more than 70 years
- Male gender
- Presence of diabetes mellitus
- Extra cardiovascular diseases
- Q waves on the ECG
- Abnormal ST-T changes not documented to be new

Treatment of ACS begins with use of aspirin, betablockers/calcium channel blockers, heparins and intravenous nitrates. Other antiplatelet agents that are available are ticlopidine and clopidogrel. GP Iib / IIIa inhibitors such as abciximab, eptifibatide and tirofiban are available for use in special situations, particularly so when cases are being taken up for percutaneous interventions (PCI). Where facilities exist for emergency PCI, it would be preferred method of treatment. The old adage, time is muscle still applies. By keeping your medical practice current, you can minimize cardiac damage and save lives.

INTERVENTIONAL CARDIOLOGY

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Cardiovascular disease is a leading cause of mortality and morbidity worldwide. Cardiology has seen spectacular advances (both in the understanding of disease processes and in the development of new treatment options) in the last half-century. Interventional cardiology is one of the major areas of development in the last three decades.

Interventional cardiology refers (by convention of usage) to catheter-based treatment of structural abnormalities of the heart and blood vessels. Pharmacological interventions, life-style modification etc., though important in their own way, are not usually considered part of interventional cardiology (notwithstanding the fact that advances in pharmacology, e.g. the development of new anti-platelet agents have had a major impact on coronary intervention, and, further, that these constitute important elements of an overall treatment program for the patient).

Interventional techniques have found widespread application in coronary artery disease, stenotic valvular lesions and peripheral vascular disease and in some forms of congenital heart disease.
Percutaneous transluminal coronary angioplasty (PTCA), commonly called " balloon angioplasty", was introduced in 1977. The procedure offered catheter-based (i.e. non- operative) relief of stenosis of the coronary arteries and rapidly developed as a treatment option for patients requiring coronary revascularization (previously possible only through bypass surgery). Technological advances enabled this technique to be applied to increasingly complex lesions and in a variety of clinical settings (including unstable coronary syndromes and acute myocardial infarction). Immediate procedural success was soon possible in over 95% of procedures, and the acute complication rate was low. There were suboptimal results in some patients and occasionally acute occlusion of the vessel (due to dissection or thrombus formation). Another major problem was the late development of recurrent narrowing in the vessel called restenosis, which occurred in about a third of patients. The development of stents (metallic meshes which scaffolded the vessel wall), and of better anti-platelet drugs, saw a significant reduction in the acute complications. Restenosis also declined significantly, though it could still occur despite stent usage. Attempts to prevent its occurrence by using radiation met with some success, but certain new problems

were also encountered. Currently, another approach (coating the stent with a drug), appears very promising. Major trials comparing angioplasty with bypass surgery in patients suitable for both procedures have reported equivalent outcomes in terms of death or infarction over medium term follow up. Recurrence of angina, repeat revascularization procedures (including crossing over to surgery) and hospitalization have been more frequent in the angioplasty patients on follow-up.

Rheumatic valvular disease remains common in India. The treatment of mitral stenosis has been revolutionized by the development of balloon valvuloplasty, which has been to shown to have results equivalent to surgery. This technique has now become the approach of choice. The high cost of the balloon, which is disposable, spurred the development of a reusable metallic dilator, offering a more cost-effective approach.

Interventional cardiology uses a variety of advanced high-technology products and this makes procedures relatively expensive. Meticulous attention to detail in all stages of decision-making is essential for optimal utilization of these procedures. In view of the high costs, durability of results is an important factor in decision-making.

Interventional cardiology definitely has an important role in the treatment of cardiovascular diseases. Since it is "high-tech"and provides "immediate" quantifiable results it has possibly been given disproportionate attention. Favoloro's statement that " ...(there is) an unreasonable gap between the medical enthusiasm devoted to acute interventions and the meager efforts currently devoted to secondary prevention" must be borne in mind to provide a sense of perspective.

NEWER ANTIBIOTICS

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Antimicrobial agents have been one of the greatest contributions to the field of Medicine. But ever since antibiotics challenged bacteria, they have developed mechanisms to resist their onslaught. Newer antibiotics are needed to treat newer pathogens, resistant bacteria, get around problems of drug allergy and interactions and also aid in sequential therapy to decrease the length of hospitalizations and the attendant expenses. The last decade has seen a significant activity in the development of newer antibiotics.

The quinolones have undergone a structural evolution since the first generation prototype of Nalidixic acid, which was effective only as a urinary antiseptic. The second generation molecules, ciprofloxacin and ofloxacin which can be used for urinary or systemic infections have given way for the third generation sparfloxacin and levofloxacin with an additional anti-staphylococcal effect and coverage for atypical pathogens and the fourth generation moxifloxacin and gatifloxacin whose spectrum includes anaerobes. The newer quinolones are effective as monotherapy for community acquired pneumonia and also have a lesser side-effect profile with regard to photosensitivity, tendonitis, gastro-intestinal and neuro-toxicity.

The macrolides have progressed from erythromycin to the newer analogues roxithromycin, clarithromycin and the azalide azithromycin. Their spectrum has expanded to include apart from gram-positive organisms, *Chlamydiae, Hemophilus ducreyi, shigella, H. pylori* and atypical mycobacterial pathogens. The newer ketolides like telithromycin are even effective against macrolide resistant streptococci. As the group evolves the drug interactions, which have been a major limitation due to the inhibition of the hepatic microsomal enzymes are also less.

As the development of resistance increases, the earlier beta-lactams have to be replaced by the glycopeptides vancomycin and teicoplanin in the management of resistant staphylocci and enterococci. These drugs are expensive, have significant toxicity and are available for parenteral use only. Currently Linezolid, an oxazolidinone and Quinupristin-dalfopristin, a streptogranin are effective against Methicillin Resistant *Staphylococcus aureus* (MRSA) and resistant enterococci.

With the explosive increase of beta lactamase producing strains of bacteria, combinations of a beta-lactam antibiotic and an inhibitor of beta lactamase have been introduced. Piperacillin with tazobactam and ticarcillin with clavulunate have joined Co-amoxyclav and ampicillin/sulbactam to help fight resistant strains.

Close on the heels of the third generation cephalosporins, which include cefotaxime, ceftazidime and cefaperazone, the latter two, having anti-pseudomonal effect, have come the fourth generation drugs. Cefepime and cefpirome have anti-pseudomonal effects and a better action against gram-negative pathogens resistant to the third generation cephalosporins. There are several second and third generation oral cephalosporins like cefuroxime, cefixime, cefpodoxime, cefdinir and ceftibuten, which are effective for sequential therapy following parenteral therapy. Outpatient management becomes feasible with these agents. Moreover cefixime is also useful in the management of multi-resistant *Salmonella typhi* infection. Monobactams like aztreonam and the carbapenems like Imepenem and Meropenem are agents with a very broad spectrum of activity. The carbepenems are active against all gram positive, gram negative and anaerobic pathogens except MRSA. Carbapenems are the drugs of choice in the management of gram-negative organisms producing extended spectrum beta lactamases. Newer aminoglycosides like Isepamicin have very good gram-negative activity and with the advent of once a day therapy, are acceptable drugs for outpatient treatment.

Other newer mycobacterial agents like Rifabutin have helped us get over the significant drug interaction and induction potential of Rifampicin especially when used with the antiretroviral protease inhibitors.

In spite of the ever-expanding armamentarium of newer antibiotics, the development of resistance in bacteria is keeping pace. This alarming trend is due to indiscriminate use of antibiotics not only for humans but also in the veterinary and farming industry. It is indeed a real possibility that we shall have untreatable bacterial infections in the not too distant future.

INFECTIONS IN INPATIENTS

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Case study

A 35 year old man has just had surgery for a perforated peptic ulcer. He now has a fever to 103F, and altered mental status. He has been on cefuroxime for the last 3 days and is not improving. Exam shows the presence of a central line, a Foley catheter and some drainage from his surgical site. His CXR shows a lower zone infiltrate.

Principles

- About 1 in 7 hospitalized patients have a nosocomial infection
- Types of infection and causative microbial flora vary significantly from community-acquired infections
- MRSA and multi-resistant Gram negative flora always need coverage
- Watch that tube!

Some general principles in treating serious infections

- Each ID problem has two components:
  - What is the syndrome?
  - What is the microbial etiology?
- Response to fever is an evaluation, not antibiotics!
- Keep in mind non-infectious causes of fever
- Get all cultures before starting antibiotics
- Always do two, not one blood culture (10 cc in aerobic set and 10 cc in anaerobic set constitutes one culture)

Empiric therapy of hospital acquired infection syndromes

- Important to decide as microbial causes of each syndrome vary
- Always decide on the syndrome before looking at the culture report
- Know local patterns of resistance
- Use adjunctive measures (surgery, drainage, etc), not just antibiotics

Percentage sensitivities at ASH

	E.coli	Klebsiella	Enterobacter	Pseudomonas
Cefuroxime	10	14	23	58
Cefotaxime	50	16	60	
Ceftizoxime	70	58	67	
Ceftazidime	38	24	53	
Cefpirome	36	56	67	

Percentage sensitivities at ASH

	E.coli	Klebsiella	Enterobacter	Pseudomonas
Cefoper-sulb	98	96	97	86
Piperacillin	28	20	53	90
Aztreonam	36	26	57	64
Ciprofloxacin	12	44	50	48
Imipenem	98	98	97	68

Percentage sensitivities at ASH

	E.coli	Klebsiella	Enterobacter	Pseudomonas
Gentamicin	26	32	43	18
Amikacin	90	78	87	66
Tobramycin	24		60	60
Netilmycin	76	58	73	54
Isepamicin	98	96	97	60

Central line infections

- Usual causes are Staphylococcus (coag pos and neg), but others too (GNB and Candida)
- Diagnosis based on positive blood culture or local purulence, not line tip cultures
- Use vancomycin for empiric therapy, but switch to beta lactams when sensitive
- Increasingly associated with endocarditis
- Remove the line, do not overwire

Urinary tract infections

- Usually caused by GNB or enterococcus
- Diagnosis based on clinical picture, not just on basis of a positive culture
- Quinolone or broad spectrum beta lactam till sensitivities back
- Remove Foley if you can
- Candida in urine resolves spontaneously with a catheter change in 30%

Surgical site infection

- <24 hrs post op: think of Streptococcus and Clostridium perfringens
- Usually secondary to Staph aureus, also bowel flora after abdominal surgery
- Always rule out deeper infection
- Surgical debridement important

Nosocomial pneumonia

- Usual pathogens are Staph aureus and GNB
- Always diagnose based on clinical features (fever, leukocytosis, purulent tracheal secretions, CXR) and then look at cultures to guide antibiotic selection
- Recent paper showed better outcome with treatment after bronchoscopic cultures
- Empiric therapy with broad spectrum penicillin or cephalosporin which has Pseudomonas and Staph coverage

Diarrhea in the hospital

- Clostridium difficile is the main pathogen
- Order a stool for C. difficile toxin assay
- If not available or patient toxic, start metronidazole orally
- Stop antibiotic if possible
- Stool culture in the hospital is unnecessary

Severe sepsis & septic shock

- Antibiotic selection based on likely microbial etiology
- If source unclear, use broad spectrum agent such as anti-pseudomonal cephalosporin, beta lactam-beta lactamase inhibitor or imipenem. Add vancomycin if Staph is likely.
- Add an aminoglycoside in high single daily dose for 1-3 days till cultures returned

Therapy of specific infections

- Open the textbook
- Use the narrowest spectrum agent that is available and effective
- Treat for appropriate time e.g. at least 14 days for most bacteremias and ventilator associated pneumonias

Antibiotic prophylaxis for clean surgery

- Clearly effective in reducing the incidence of surgical site infections
- However prolonged antibiotic use results in side effects, super-infections, increased cost and ultimately antibiotic resistance
- Antibiotics have to be in the system at time of incision and for duration of surgery.
- No role for antibiotics after day of surgery

Antibiotic selection for surgical prophylaxis

- Based on anticipated flora on skin at time of incision e.g Staph aureus over limbs, chest
- Examples include cefazolin for above and cefotetan for abdominal surgery
- No role for aminoglycosides in general
- Usually hospital infection control committee and appropriate specialists confer and decide on which antibiotic for each procedure

RAY OF HOPE IN DERMATOLOGY

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Sunlight as the ultimate physical source of heat, light, energy and thus life on earth has been revered in religious beliefs. Ancient Greeks, Egyptians, Persians and Indians all worshipped the sun. The word radiation has been derived from 'Ra' denoting the Egyptian sun god. Herodotus has recorded the healing effect of the rays of the sun as early as in the 2nd century. It's beneficial effects on rickets were later established in the 18th century.

Sunlight consists of several types of energy including cosmic rays, gamma rays, x-rays, UV rays, visible light, infrared rays and radio rays. For the skin UV-light and visible light play a major role inducing changes.

Phototherapy

The use of UV light for treating diseases is called phototherapy. Dermatologists use only the UV spectrum, which consists of photons of wave length ranging from 200 to 400 nm in length. UV has UVA, UVB and UVC. UVC is germicidal and is not used in therapy. Only UVA and UVB are used in phototherapy of skin diseases.

UV radiation appears to inhibit DNA synthesis, to modulate antigen-producing cells and to stimulate melanocyte transfer from adjacent cells. Thus phototherapy is useful in diseases like,

Vitiligo	-	where there is melanocyte destruction
Psoriasis	-	where there is increased epidermal turn over
Atopic eczema		
Cutaneous T cell lymphomas	-	where there is alteration in the immune system

Photo Chemotherapy or PUVA

While UVA light alone has some influence on diseases like psoriasis, it becomes a most effective treatment when combined with Psoralen – a photosensitizing chemical. It is a furocoumarin found in many plants.

The use of Psoralen from seeds of plants like Ammi majus and subsequent sun exposure, to treat diseases like vitiligo has been recorded in the Atharva Veda. This therapy is called as PUVA therapy where P stands for Psoralen and UVA for ultraviolet A radiation.

Need for phototherapy unit

Though sunlight, the most obvious source of UVA, is available in plenty in our country, it has certain inherent disadvantages. The amount of sunlight available varies with the time of day, season and there is also lack of privacy. These have been overcome in modern medicine by using artificial sources of sunlight like, fluorescent tube lights, which emit either UVA or UVB. Phototherapy equipments are now available. These are upright booths lined on all sides by fluorescent tube lights. Patients can thus have treatment in absolute privacy and at a time of their convenience.

Methodology of PUVA

Before starting a patient on PUVA baseline investigations including liver & kidney function test, hemogram & ophthalmic examination are done. Those with a history of liver, kidney, photosensitive diseases and skin malignancies are excluded from this therapy. Similarly those who had radiation therapy or those who are on photosensitive drugs, pregnant women and children below 10 years are also excluded.

Psoralen in given in doses of 0.6mg/kg, with a small meal, followed by UVA exposure, after 1 & ½ hrs which is the time taken for the drug to reach the skin. Patient has to wear protective goggles before, during and 8 hrs after treatment.

He stands in the phototherapy unit, wearing protective clothing over sensitive areas like genitalia. The time spent in the unit is increased gradually with each visit. During every visit the UVA dosage, time of exposure, total cumulative UVA dosage and total number of treatments are recorded. It takes about 20-30 treatments to treat psoriasis and nearly a 100 to cause atleast 75% repigmentation in vitiligo.

Side effects

Short-term complications include sunburn like reactions, which can be treated with mild sunscreens and emollients. Taking the drug with food can minimize nausea.

Long-term side effects include dryness of skin, hyper pigmentation, increased ageing/wrinkling of skin, increased pre cancerous skin conditions, rarely, SCC's and recently, melanomas have been reported after more than 250 treatments. But with good monitoring none of these side effects will occur.

Conclusion

In our 2 years experience we have seen only mild sunburn like reactions. We also take care to see that we do not exceed 250 treatments in order to avoid long-term side effects. The quality of life of many a psoriatic patient has dramatically improved with PUVA. Similarly, the lives of patients with vitiligo have become rosier after the disappearance of the unsightly depigmented patches following PUVA.

Phototherapy has indeed proved to be "a ray of hope" or should I say "rays of hope" in the field of Dermatology.

DENTISTRY IN THE 21ST CENTURY

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In this century, science and technology have provided the necessary basics that have propelled dentistry into fields unimaginable only a short time ago. This allows dentists to offer various modalities that can aid and improve diagnostic and clinical techniques.

The growth of Aesthetic Dentistry and the cosmetic demands of our patients call for sophistication in dental diagnostics, communication and fabrication of the end product. Preventive dentistry is now greatly reducing dental morbidity for dental disease account for more pain, sufferings and loss of working hours than almost any other disease. Although, dental caries is the most widely spread chronic disease affecting the world's population today, it is gum disease – or periodontal disease that gives rise to tooth loss. It has been clearly established that bacteria is essential in causing periodontal disease. The three most important factors determined to date are

1. Smoking
2. Compliance with oral care regimens and oral hygiene
3. Genetic susceptibility

1. Smoking has been established as a major risk factor in affecting a patient's response to bacterial plaque. It has a more dominant effect on attachment and tooth loss than poor compliance and severe gingival inflammation.A study which involved analyzing data from the III National Health and Nutrition Examination survey from 1988 to 1994 revealed a 50-60% increased risk of periodontal disease among non-smokers who are around smokers than those who are not.

Active smoking is an important risk factor for periodontal disease, one of the many chemicals that gets into the body when a person actively smokes or breathes someone else's smoke is nicotine. Earlier studies have shown that nicotine in cigarette smoke impairs the immune system and causes blood vessels to constrict including blood vessels in the tissues around the teeth. A decrease in oxygen along with an impaired immune system response creates a favorable environment for bacteria that causes periodontal disease.

2. Unless bacterial plaque is routinely removed by practicing compliance with oral care regimens and oral hygiene, immature bacteria accumulate on teeth, and plaque continues to grow until it contains harmful

pathogens that contribute to the destruction of tissue and the supporting tooth structures. Oral hygiene and compliance are behavioral factors, which implies the ability to control and or modify their use.

3.Individuals with genetic susceptibility are more likely affected by poor oral hygiene/compliance and smoking, leading to severe periodontal disease and tooth loss. Genetically susceptible individuals are those who produce more interleukin 1(IL-1), an inflammatory mediator, in response to bacterial plaque, resulting in rapid and extensive damage to the surrounding tissues.

A study by Kornman & Colleagues found that 67% of patients with severe periodontal disease had the IL-1 genotype. Looking at these factors, and considering each patients risk cost – effective planning and treatment can be provided.

Chronic periodontal disease may contribute to diabetes, according to a review of recent research, while it has been established that people with diabetes are more prone to developing periodontal disease, new research is suggesting that periodontal disease may, in turn, be a risk factor for diabetes. Systemic antibiotics are recommended to control chronic gram-negative infection as part of periodontal therapy for all diabetic patients with periodontitis.

Scientists for the first time, have offered the first direct physical evidence that a deficiency in white blood cell function can lead to early – onset periodontal disease, or aggressive periodontitis. The conclusion comes from studies of genetically engineered mice that are unable to fight periodontal infections.

Elevated C-reactive protein may explain MI & Periodontal disease link - Patients with severe form of periodontal disease have elevated levels of c-reactive protein compared to patients with out periodontal disease, according to Dr. Ernesto De Nardin – Asst. Prof, of microbiology and oral biology at the University of Buffalo, New York school of Medicine & Dentistry.

As bacterial scores arte reduced and disease arrest is observed patients will move to the low caries – risk category. For the moderate – to high risk patients, restoration materials that contain fluoride are formed if they are appropriate for that patient.

Topical fluoride is an effective agent for remineralization and many difficult fluoride products are available including varnishes, gels, raises, dentrifices and fluoride - re leasing restoration materials. Chlorhexidine products may be required to reduce streptococcus mutans scores prior to treatment for high-risk patients. In a study performed with patients suffering from radiation-induced caries, weekly doses of chlorhexidine and sodium fluoride resulted in the remineralization of initial carious lesions. It is recommended that active lesions be treated with a daily one –minute rinse of 0.12% chlorhexidine gluconate for 4-6 weeks.

The use of dental amalgam as a direct restorative material has been a subject of controversy for the past two decades. At the present time amalgam is still being widely used for its cost durability and ease of manipulation make it the number one choice for restoring posterior teeth. In response to concerns about mercury, demand for amalgam alternatives has increased, although no controlled studies have been published demonstrating systemic adverse effects from amalgam restorations.

Recently, a pair of Japanese researchers said that amalgam causes the chronic skin disease atopic dermatitis and other allergic dermatitis. After replacing the amalgam, approximately 70% of the patients saw improvements in their dermatitis condition a year later, and about 58 % completely recovered. Out of 300 patients, tested for lymphocytes allergic reaction to heavy metals such a s mercury, 98% of patients tested positive for a mercury allergy. In most cases, it is recommended that amalgam filling not be used in young children and pregnant women.

Composites

Composite restorations are slowly replacing amalgam as the filing material of choice.

Posterior composite is not at this time a total replacement for amalgam as a restorative material but such a time is quickly coming

Reasons : Packable resin – based composite

Good handling characteristics

Stimulate natural tooth structure - possible to deliver restoration that is truly natural in appearance

Wear resistance

Techniques are simplified (no complex cavity preparation needed)

Main disadvantages are lack of strength and decreased abrasion under resistance, which is still not comparable to silver amalgam

Root Canal treatment or Endodontics

With the advent of this procedure, tooth extraction is becoming a thing if the past broken down, decayed and infected teeth can be saved now. Endodontics involves removing the inflamed or infected pulp, cleaning the empty nerve channel and filling the root canal. Endodontic therapy is still found to be safe and effective compared to extraction for controlling the risk of bacteremia especially in patients with cardiac problems.

Prosthodontics is concerned with replacement of missing teeth and contiguous structures of the oral cavity and the face. The various techniques of replacement include the fixed type and removable types. The removable prostheses also called as removable dentures are indicated for extensive replacement of teeth

and older age and where cost is a major consideration. The fixed replacements are more precise, sophisticated and expensive, but they also require preparation or grinding of teeth present for support.

Dental implants have begun to play a greater role in tooth replacement but the cost is still prohibitive. The advantage of implants lie in preservation of alveolar bone and no preparation of teeth. Even complete dentures especially the lower ones which are unstable can be supported by dental implants making them fixed.

Prosthodontic rehabilitation also includes replacement of teeth and associated structures for patients affected by congenital deformities like cleft lip and palate by providing feeding plates and obturators.

Artificial replacement of eyes, ears and nose and parts of face, where reconstruction may not be carried out, also falls under this preview. Fixed replacements for children and adolescents, where conventional replacements cannot be done may be done with the newer resin bonded prostheses.

Orthodontics means – straightening of teeth. Orthodontics involves different types of appliances like

- Removable
- Functional
- Orthopedic &
- Fixed (braces) appliances

Early treatment is instituted in children to correct existing or developing skeletal, dental and muscular imbalances. By initiating orthodontic and orthopedic therapy at a younger age, the need for complicated orthodontic treatment involving extractions of teeth and orthogenic surgery is minimized.Orthodontics ensures proper dental alignment especially facial balance in children, adolescents and even adults.

Latest in orthodontics

- Invisible braces for adults (ceramic)
- Twin block functional appliances for children with retrognathic mandible
- Headgears therapy before and after treatment
- PEA (Pre-adjusted edgewise appliances)

Developmental deformities of the jaws

Developed deformities of the jaws are those deformities that present as malocclusion of the teeth, malrelation of the jaws and associated facial disfigurement. They are thought of most as congenital in origin, but they may result from other causes. **Orthognathic surgery** aims at correcting jaw and facial deformities surgically and usually in combination with orthodontic appliances either before or after

surgery. The surgical correction of these deformities is one of the most challenging and intriguing aspects of oral surgery. Helping persons so afflicted is one of the most gratifying services that is possible to render.

Causes of facial deformity:

1. Hereditary factors which determine the pattern of facial growth
2. Injuries or infections of the jaws/face during infancy or childhood

Problems associated with abnormal jaw / facial growth

1. Poor facial appearance
2. Eating difficulties

Diagnosis and treatment planning

Diagnosis of jaw deformities is usually by clinical examination and special records like X-rays (lateral view of face), photographs, and plaster models of the teeth and jaw. Deformities of the jaws are corrected by the standard Le-fort osteotomy and mandibular osteotomes.

Modern anaesthesia and drug therapy, refined surgical techniques, improved instrumentation and the combination of skills of the oral surgeon and orthodontist have meant that almost every dentofacial deformity can be now corrected with improved functional, aesthetic and psychological benefits.

A future without drills and fillings not far away

Cosmetic laser dentistry – a new area employing advanced technology to produce sculpted cosmetic results. Patients benefit by experiencing diminished or virtually no bleeding, swelling or pain after a laser procedure. Some of the applications are for frenectomy, implant uncovering, surgical exposures, treatment of draining fistulas and aphthous ulcers, coagulating and decontaminating extraction sites, excisional biopsies and much more.

More and more patient come with problems such as – teeth that are chipped, fractured, worn or discolored or diastemas that are too wide. They want to be able to smile again, without embarrassment or discomfort. In increasing numbers, these patients are giving not only a new smile, but also a vastly improved self image and social well-being.

Imagine a visit to the dentist where cavities and gum disease can be prevented by using gene therapy. Imagine being able to repair or regenerate your patients teeth using their own DNA. Instead of filling, a dentist will someday modify the specific bacteria in a person that cause dental disease in the first place. Simple swab from inside the mouth will provide enough DNA to develop individualized dental treatment in the future. As health professionals, we are committed to prevention. And with all the advances, in recent years, prevention of dental disease is now very possible.

CEREBRAL PALSY – ONE STEP AT A TIME

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Cerebral palsy is a name given to a group of conditions in which there is a disorder of movements and posture caused by damage to the brain. This may be caused by a developmental abnormality or an injury to the brain occurring during delivery or shortly after birth. This condition is non progressive but permanent.

The incidence in the western world is 1 –2 per thousand live births .In addition to the motor deficit, some of these children may have other dysfunctions such as, mental retardation, hearing, visual, or speech problems.

The different presentations of cerebral palsy are as follows:

1) Hemiplegia - One-sided involvement.
2) Diplegia – Lower limbs affected more than the upper limbs
3) Quadriplegia – All four limbs involved.
4) Ataxia – Balance disorder.
5) Athetoid – Involuntary movements.

Akash Centre for Cerebral Palsy

When faced with a situation as such, parents go from doctor to doctor in order to do the best for their child. Most often they are suggested physiotherapy. As the results of therapy are slow, therapists are changed frequently.

At Akash we have tried to overcome these problems in order to give the child quality medical care, thereby giving the child a chance to independence. This is achieved in the following manner:

Step 1

The usual complaint of the parents of the children with cerebral palsy is delayed milestones. An evaluation of the child is done which includes a detailed history, physical and neurological examinations by a team of medical and paramedical professionals.

Step 2

Once the diagnosis and type of cerebral palsy are confirmed a treatment plan is formulated for the individual child. The goal of the plan would be to maximize function and minimize the development of secondary problems such as joint contractures. The problem areas for the child would be looked into and a long-term plan defined.

Step 3

The corner stone of treatment is physiotherapy. A comprehensive set of exercises would be tailored for the child. This would involve exercises for movement, sitting, hand control and speech. Daily activities as toilet training and feeding would also be looked into. These insecure children are motivated to doing simple tasks thereby developing their personality. A cheerful atmosphere is created whereby these children enjoy the therapy session.

Step 4

Periodically the team would evaluate the child's progress and counsel the parents. Sometimes it may be necessary to use mobility-aiding orthoses or resort to surgery to correct joint contractures. Selective use of spasticity reducing injections (Botox) could be used to improve mobility.

SMF INITIATIVE

This SMF initiative is a non-profit community service, to give evidence based, quality care for these special children. Goal oriented, sustained physiotherapy in a cheerful environment, would motivate these children to mobilize and thereby develop their personality.

INTAKE – OUTPUT CHARTING

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Nothing is more difficult to obtain in a modern hospital than an accurate record of the intake and output! The I/O chart helps in giving an idea of the fluid and electrolyte balance of the patient and to plan correction of any deficits.

The Intake record

A typical intake chart records the type, amount & route of administration with respect to time. The usual routes of administration are: oral, intravenous, nasogastric, blood and blood products. A record of solid food intake is important in very young children. Semisolid foods- e.g. gelatin/ice-cream are recorded as fluids. For calculating intake of ice chips: The amount of chips/2 in ml of water is taken as the intake.

The Output record:

Urinary output: The time, amount and colour of each voiding are recorded. If renal function is a major concern, urinary catheterisation and recording of hourly output is performed. It is important to ensure that urine is not discarded before measurement.

Wound drainage: All drainage from body orifices or artificial openings are to be recorded accurately. If there is excessive discharge from the wound, dressings have to be weighed in order to get an accurate idea of the output. Fluid loss is calculated as the Wet wt – Dry wt.

Gastrointestinal losses: GI losses may be through vomitus, GI drainage or stools. Colour, consistency and odour are to be noted. With respect to nasogastric irrigation, the amount of fluid retained during irrigation is added to the intake and needs to be subtracted from total drainage. With respect to stools, the consistency and number of stools are noted.

Other output: This may be aspirated fluid like thoracentesis or paracentesis, diaphoresis and perspiration. Diaphoresis is difficult to measure and when required, special laboratory equipment are needed. Similarly perspiration is difficult quantify, and when required, dry and wet weight of clothing are to be calculated.

The Fluid balance

The importance of charting the intake and output lies in its utility to give the fluid balance at the end of the day. A positive balance implies that intake has been more than output and a negative balance the reverse.

FLUID THERAPY IN CHILDREN

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About 80% of body weight is water in early infancy and it decreases to 60-65% by the end of first year and it is fairly constant thereafter. Young infants due to renal tubular concentration immaturity are vulnerable to water loss. High metabolic rate and larger surface area compared to total body in young infants favour rapid fluids loss. An young infant exchanges roughly one-half of his or her ECF everyday compared to one-seventh in an adult. Thirst mechanism is less effective in young infants.

Water requirements in children

Body wt.	Volume/amount in 24 hrs.	Volume per hour
< 10 kg	100 ml/kg	4 ml/kg/hr
11 – 20 kg	1000 ml + 50 ml/kg for each kg > 10 kg	40 ml/hr + 2 ml/hr per kg > 10 kg
> 20 kg	1500 ml + 20 ml/kg for each kg > 20 kg	60 ml/hr + 1 ml/hr per kg > 20 kg

- Add or subtract 12% for each degree centigrade above or below temperature of 37.8°C
- In neonates the fluid needs are far less in the first week of life, 60-65 ml/kg in the first 2-3 days of life is adequate and gradually increased to 100 ml/kg after a week.

Normal electrolyte and glucose requirements

Sodium	3 – 4 mEq/kg/day
Potassium	2 – 3 mEq/kg/day
Chloride	3 – 4 mEq/kg/day
Calcium	50 – 200 mg/kg/day
Magnesium	0.3 – 0.5 mEq/kg/day
Glucose	3 – 6 mg/kg/mt

Points to remember

- Any rehydration fluid must contain 5% Dextrose except in DM where it has to be decided on need.
- 10% glucose in water will be ideal in neonates
- Neonates usually do not need Na^+ or K^+ in the first 48 hours except in the presence of continued loss from GIT
- Any continued loss of Na^+ must be replaced adequately.
- Correction of hyponatremia: Na^+ required is (135 – X) x 0.6 x wt. in kg where X is the actual sodium observed.
- In symptomatic hyponatremia use hypertonic saline (3% NaCl provides 0.5 mEq/ml) at the rate of 12 ml/kg/hr. Initial correction is upto 125 mEq/L of Na^+. If child is convulsing rapid infusion of 3 ml/kg over 10-15 minutes can be given. If acidosis worsens after acute correction replace part of sodium (nearly ¼th of Na deficit) as sodium bicarbonate (0.9 mEq/ml). Subsequent elevation of sodium must be done with small increments.
- Aysmptomatic hyponatremia can be treated with 5% GNS (15 mEq/100 ml) separately or in pediatric maintenance solution if urine output is good.
- In hypertonic dehydration 5% GDW with 25 mEq/L of sodium will be adequate. Sodium free solution is likely to precipitate seizures.
- For hypokalemia IVF should contain 4 mEq/100 ml of K^+ in the fluid given in common setup. In ICU setup 6-8 mEq/100 ml of K^+ can be tried with monitoring. Usually not more than 4 mEq/kg can be given in the course of 24 hours.
- Bicarbonate deficiency is usually corrected if the value is less than 15 mEq/L in the presence of metabolic acidosis.
- Disturbed metabolism of fluid and electrolytes in various clinical states other than diarrhoeal situations should be based on the cause of disturbance like renal, CNS and respiratory causes.

Treatment of dehydration in diarrhoea

Mild dehydration ($\leq$ 5% deficit)

- Give oral fluids (home available fluids) more than usual.
- ORS can be given
- IV fluids are not usually required
- Watch for ongoing loss and increasing dehydration.

Moderate dehydration (5-10% deficit)

- Continue breast feeds
- 75 ml/kg of ORS over 4 hours orally or via NG tube
- If clinical condition worsens (increase in copious watery stools and signs of dehydration more marked) then RL 75-100 ml/kg over 3 – 4 hours.
- Continue ORS for stool replacement and oral feeds.

Severe dehydration

- Infants:

  30 ml/kg RL in first hour

  70 ml/kg RL in next 5 hours

  Older children:

  30 ml/kg RL in 30 minutes

  70 ml/kg RL in next 2.5 hours

  Repeat second bolus of 30 ml/kg if signs of dehydration persists after first bolus.

- If child is able to drink commence oral fluids. In some, maintenance IV fluids (Isolyte P) may be required along with stool losses with ORS

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FLUID RESUSCITATION IN TRAUMA

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Introduction

The incidence of trauma is on the ascent. With the existing poor prehospital support system, the management of trauma victim is entirely in our hands.

Death in accident victims typically shows trimodal distribution. The *first peak* of death is within few minutes of accident. The *second peak* occurs in the initial few hours. Deaths are due to intracranial hematomas, hemopneumothorax, spleen / liver injuries or other multiple injuries with significant blood loss. The aggressive resuscitation during the initial few hours is of paramount importance in the care of trauma victims. The *third peak* occurring several days or weeks after the initial injury is due to sepsis and multiple organ failure. Care provided during the initial hours has a direct effect on late morbidity and mortality.

Primary survey

In the severely injured patient, the vital functions must be assessed quickly and simultaneously logical sequential treatment priorities must be established. This process constitutes the ABCs of trauma care and identifies life-threatening conditions.

A – Airway maintenance with cervical spine control.

B – Breathing and ventilation.

C – Circulation with hemorrhage control.

D – Disability – Neurostatus.

E – Exposure

In this article we focus on resuscitation of shock (circulatory management).

Resuscitation of traumatic shock

Recognition of shock

Full-blown shock as evidenced by low blood pressure and inadequate perfusion of the skin, kidney & central nervous system is easy to recognize. Sole reliance on blood pressure as an indicator of shock results in delayed recognition of shock, because compensatory mechanisms of the sympathetic system preclude measurable fall in blood pressure until patient has lost up to 30% of circulatory volume. Hence specific attention should be directed to heart rate, respiratory rate, skin circulation, pulse pressure, orthostatic hypotension & neurostatus. No laboratory tests immediately diagnose shock.

Classification

Class I haemorrhage: Loss at upto 15% at blood volume. Clinical symptoms and signs are minimal:

Class II haemorrhage: 15 to 30% blood loss: Manifestations include tachycardia, tachypnea, decrease in pulse preserve and orthostatic hypotension:

Class III haemorrhage: 30 to 40% blood loss: They present with the signs of inadequate perfusion, which includes marked tachycardia and tachypnea, hypotension and altered mental status.

Class IV haemorrhage: > 40% blood loss: They present with profound hypoperfusion picture: Thready pulse; very low BP, cold and clammy skin, and marked by depressed mental status.

Vascular access:

It must be obtained promptly; Establish two large bore (preferably 16G/Grey color) peripheral intravenous cannulae immediately. The most desirable sites for peripheral IV line in adults are the forearm or antecubital veins. Long central venous catheter is inferior to large short peripheral cannula.

Initial fluid therapy:

Isotonic electrolytes solutions (Normal saline and Ringer's lactate) are the initial choice: 5% Dextrose water should not be used in resuscitation. Colloids like Gelatin, starch or albumin have not shown survival benefit in clinical trials when compared with crystalloids (colloids have theoretical superiority over crystalloids).

Class I & II shocks requires only crystalloids and class III & IV shocks requires blood in addition to crystalloids. Fluids should be given as a bolus as rapidly start with 10ml/kg as bolus and repeat with close monitoring at vitals, saturation with pulse oximety and careful auscultation of wings for basal crackles between boluses as possible. In adults upto 30 to 40ml/kg can be given rapidly. (In a 70kg man about 2.5L). Further fluids are based on the response. The return at normal blood pressure, pulse rate and pulse pressure are positive signs that circulation is normalizing.

Blood is required for Class III and IV shock victims. The main purpose in transfusing blood is to restore the oxygen carrying capacity at the intravascular volume, while volume resuscitation can be accomplished by crystalloids, type specific blood (uncross matched) is the first choice for patients with life threatening shock. It type specific blood is unavailable; 'O' negative blood can be used.

Pitfalls

1. Not supporting ABCs, and concentrating on wound management.
2. Equating BP with adequate perfusion
3. Using Dextose fluids for resuscitation
4. Initiating Inotropes / vasopressors without replacing fluid and blood.

Conclusion

Aggressive resuscitation with fluids in traumatic shock reduces the morbidity and mortality in trauma victims.

ADENOTONSILLITIS – WHEN DO WE OPERATE?

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Adenotonsillitis though a common entity in clinical practice, has always been plagued by misconceptions regarding the role of surgery. A basic knowledge of Adenoids and Tonsils is the first step towards better understanding of adenotonsillitis.

Anatomy and Physiology

Adenoid and tonsils are lymphoid organs situated in the nasopharynx and oropharynx respectively. The adenoids are situated in the nasopharynx, related posteromedially to the Eustachian tube predisposing to recurrent attacks of otitis media in children with enlarged and infected adenoids. Commonly the adenoids regress by the age of 5-6 years and the tonsils by the age of 12 years. X-ray skull lateral view and diagnostic nasal endoscopy, and outpatient procedure, will clearly show the size and amount of nasal obstruction.

Clinical Presentation

Adenotonsillitis is a common disorder in children and it is unusual for a child not to have one or two episodes of adenotonsillitis. During an acute episode of adenoiditis the clinical spectrum is fever, mouth breathing, rhinitis and ear pain. Acute Tonsillitis, seen in all age groups, the common presenting complaints are painful swallowing, recurrent sore throat, and fever with constitutional symptoms. Clinical examination reveals hypertrophied tonsils, pus or infected secretions in the crypts with bilateral jugulodigastric nodes. Infection resolves with conservative treatment alone in majority of the cases without complication.

Indications for Surgery

Several factors need to be considered prior to Adenotonsillectomy. Indications for surgery, need to be vigorously examined. Size of tonsils, assessment of nodes, bacteriological and serological examination is not reliable in deciding the surgery.

Only absolute indications have been discussed which are as follows:

1. Recurrent episodes of acute tonsillitis – 6 or more attacks per year for at least 2 years.
2. Single attack of Peri-Tonsillar Abscess.
3. Obstructive sleep apnoea.
4. Localised disease of tonsils like tonsilolith, tonsillar cyst, and imbedded foreign body.
5. Carcinoma tonsil, excision biopsy in suspected case of Hodgkins lymphoma.
6. Tonsil acting as source of infection (Rheumatic heart disease, glomerulonephritis, Scleritis). Positive bacteriological report on Group A Beta haemolytic Streptococcal and those not prepared for antibiotic prophylaxis.

Adenoidectomy alone is indicated in cases of

1. Enlarged adenoids obstructing the airway.
2. Recurrent otitis media and unresolving effusion associated with enlarged adenoids.
3. Mouth breathing.

Patients presenting to the primary care physicians with recurrent infections fulfilling the above criteria need to be evaluated by ENT specialist.

PREOPERATIVE DIABETIC CONTROL

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Common Principles

1. Should be done in co-ordination with anaesthetist, surgeon and physician and should also assess complications if any.
2. Though optimal control is ideal and desirable, it may not always be feasible, especially in emergency situations.
3. Long acting drugs both insulins and OHA's are best avoided on the night prior to surgery.
4. Some patients will need glucose-insulin infusions peri-operatively

Elective surgery

In surgeries not requiring overnight fasting, OHA /insulin are omitted on the morning of the procedure and restarted after resumption of oral diet in patients who are well-controlled . In poorly controlled patients, an attempt must be made to achieve good control. However surgery should not be indefinitely delayed.In such patients,it may be necessary to admit for a few days to achieve glycemic control.

Emergency surgery

In poorly controlled patients,

1. Emergency surgery should NOT be delayed on account of sugars.
2. The risk-benefit should be explained to the patient.
3. Sugars should be controlled with insulin,with infusions if necessary.
4. Electrolytes ,fluids and acid-base levels may need monitoring.
5. ICU/IMCU care may be needed in the post-op period.

Impaired glucose tolerance (igt) & impaired fasting glucose (ifg)

IGT is defined as 2hour plasma venous glucose value of 140-199 mg /dl after 75 gm oral glucose. This is the WHO definition.

IFG is defined as Fasting plasma venous glucose value of 110-125mg/dl. This is the ADA definition.

A large number of patients with these conditions eventually develop overt diabetes. IGT is an independent risk factor for macro-vascular disease. While cut-off points are no doubt useful clinically it should also be kept in mind that vascular complications do develop across an entire range of values. Reduction to the most physiologically acceptable levels translates into lesser complications. These conditions (IGT&IFG) represent a window of opportunity to prevent progression to overt Diabetes.

DYSLIPIDEMIA

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STEP 1

Determine lipoprotein levels – obtain complete lipoprotein profile after 9- to 12-hour fast.

ATP III Classification of LDL, Total, and HDL Cholesterol (mg/dL)

LDL Cholesterol – Primary Target of Therapy

<100 Optimal

100-129 Near optimal/above optimal

130-159 Borderline high

160-189 High

>190 Very high

Total Cholesterol

<200 Desirable

200-239 Borderline high

>240 High

HDL Cholesterol

<40 Low

>60 High

STEP 2

Identify presence of clinical atherosclerotic disease that confers high risk for coronary heart disease (CHD) events (CHD risk equivalent):

- Clinical CHD
- Symptomatic carotid artery disease
- Peripheral arterial disease
- Abdominal aortic aneurysm.

STEP 3

Determine presence of major risk factors (other than LDL):

Major Risk Factors (Exclusive of LDL Cholesterol) That Modify LDL Goals

Cigarette smoking

Hypertension (BP >140/90 mmHg or on antihypertensive medication)

Low HDL cholesterol (<40 mg/dL)*

Family history of premature CHD (CHD in male first degree relative <55 years;

CHD in female first degree relative <65 years)

Age (men >45 years; women >55 years)

STEP 4

If 2+ risk factors (other than LDL) are present without CHD or CHD risk equivalent, assess 10-year (short-term) CHD .

Three levels of 10-year risk:

- >20% — CHD risk equivalent
- 10-20%
- <10%

STEP 5

Determine risk category:

- Establish LDL goal of therapy
- Determine need for therapeutic lifestyle changes (TLC)
- Determine level for drug consideration

STEP 6

Consider adding drug therapy if LDL exceeds levels shown in Step 5 table:

- Consider drug simultaneously with TLC for CHD and CHD equivalents
- Consider adding drug to TLC after 3 months for other risk categories.

STEP 7

Consider adding drug therapy if LDL exceeds levels shown in Step 5 table:

- Consider drug simultaneously with TLC for CHD and CHD equivalents
- Consider adding drug to TLC after 3 months for other risk categories.

Treatment of the metabolic syndrome

- Treat underlying causes (overweight/obesity and physical inactivity):
  - Intensify weight management
  - Increase physical activity.
- Treat lipid and non-lipid risk factors if they persist despite these lifestyle therapies:
  - Treat hypertension
  - Use aspirin for CHD patients to reduce prothrombotic state
  - Treat elevated triglycerides and/or low HDL (as shown in Step 9).

STEP 8

Identify metabolic syndrome and treat. Clinical detection of the metabolic syndrome depends on the presence of any three of the following.

- Abdominal obesity
- Raised triglycerides
- Low HDL
- Hypertension
- Impaired fasting glucose

STEP 9

Treat elevated triglycerides.

ATP III Classification of Serum Triglycerides (mg/dL)

<150 Normal

150-199 Borderline high

200-499 High

³500 Very high

Treatment of elevated triglycerides □³150 mg/dL)

- Primary aim of therapy is to reach LDL goal
- Intensify weight management
- Increase physical activity
- If triglycerides are >200 mg/dL after LDL goal is reached, set secondary goal for non-HDL cholesterol (total – HDL) 30 mg/dL higher than LDL goal.

If triglycerides 200-499 mg/dL after LDL goal is reached, consider adding drug if needed to reach non-HDL goal:

- Intensify therapy with LDL-lowering drug, or
- Add nicotinic acid or fibrate to further lower VLDL.

If triglycerides >500 mg/dL, first lower triglycerides to prevent pancreatitis:

- Very low-fat diet (<15% of calories from fat)
- Weight management and physical activity
- Fibrate or nicotinic acid
- When triglycerides <500 mg/dL, turn to LDL-lowering therapy.

Treatment of low HDL cholesterol (<40 mg/dL)

- First reach LDL goal, then:
- Intensify weight management and increase physical activity
- If triglycerides 200-499 mg/dL, achieve non-HDL goal
- If triglycerides <200 mg/dL (isolated low HDL) in CHD or CHD equivalent consider nicotinic acid or fibrate.

OSTEOPOROSIS: PRACTICE GUIDELINES

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Osteoporosis is a major health problem affecting women and men in our country. Preservation of bone health is a major part of the agenda in preventive medicine. With the increase in longevity of men and women the occurrence of osteoporotic fractures have become a major source of morbidity and mortality in the elderly. However availability of bone densitometry and newer therapeutic agents have made the diagnosis and treatment of osteoporosis easier.

The NIH consensus of 2000 has addressed the following key questions.

1. What is osteoporosis and what are its consequences?
2. How do risks vary among different segments of the population?
3. What factors are involved in building and maintaining skeletal health throughout life?
4. What is the optimal evaluation and treatment of osteoporosis and fractures?

1. What is osteoporosis and what are its consequences?

Osteoporosis is defined as a skeletal disorder characterized by low bone mass and micro architectural deterioration of the bone with consequent increase in fragility and fractures. Primary osteoporosis can occur in both genders in all ages but often follows menopause in women and occurs later in life in men. Secondary Osteoporosis is result of medications, other medical conditions. Consequences of osteoporosis are fractures, pain due to fractures, increased mortality and morbidity with hip fractures. Other systems may be affected and the financial and psychosocial burdens can be disabling.

2. How do risks vary among different segments of the population?

Female sex, racial differences in bone mass, advanced age, post menopausal state, low body weight and BMI, family history, smoking and prior fracture are the major risk factors. Peak bone mass attained is a crucial determinant of osteoporosis risk. Hypogonadism, genetic and endocrine disorders, GI diseases, hematological diseases, nutritional deficiencies, drugs and host of other conditions like CHF, end stage renal disease and alcoholism may be associated with secondary osteoporosis.

3. **What factors are involved in building and maintaining skeletal health throughout life?**

Peak bone mass is attained as early as early 3rd decade. Adequate calories, calcium and vitamin D intake, avoidance of excess protein, caffeine, phosphorus, and sodium are the nutritional factors involved in bone remodeling. Regular physical exercise improves muscle mass. bone density and quality of life. Gonadal steroids, growth hormone and IGF I play a crucial role in acquisition and maintenance of bone mass.

4. **What is the optimal evaluation and treatment of osteoporosis and fractures?**

These include the following

- Complete history and physical exam
- Calcium, albumin and alkaline phosphatase to rule out osteomalacia.
- Bone mineral density estimation
- Bone turnover markers such as urinary pyridinolines and deoxy pyridinolines and serum and urine levels of type I collagen telopeptides (CTX and NTX) which are indices of bone resorption and bone specific alkaline phosphatase and osteocalin which are indices of bone formation are available to make quick assessment of bone remodeling.

5. **Who should be evaluated?**

Individualized approach is recommended; a bone density measurement should be considered when it would help the patient decide whether to institute treatment to prevent osteoporotic fracture.

6. **What are the effective medical treatments?**

- Adequate calcium (1000-1500mg/day) and vitamin D (400-800 IU/day) intake
- Physical activity
- Bisphosphonates like alendronate, Risedronate, Pamidronate and Ibandronate
- Hormone replacement therapy
- Selective Estrogen Reception Modulators
- Parathyroid hormone
- Calcitonin - Nasal SC

7. **What are the areas for future research?**

- Strategies for maximizing bone mass
- Identification and targeting specific genetic factors predisposing to osteoporosis
- Novel approaches for stimulating bone formation in glucocorticoid induced osteoporosis.
- More accurate fracture risk assessment

- Characterize and validate quality of life tools
- Information regarding screening guidelines
- Research into relationship between neuro psychiatric disorders and osteoporosis
- Effect of combination therapy
- Optimal evaluation and management of osteoporosis
- Development and assessment of anabolic agents
- Public education
- Emphasizing the role of nutrition, dietary supplements, micronutrients.

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For 7 year Rajan his trauma with cancer was further complicated when he was initially diagnosed for T.B. Only later when he was brought to the Institute of Child Health he was diagnosed with Hodgkins's disease. Rajan's father an agricultural laborer earning just enough to keep his family going was not sure how he would meet the medical expenses of his son. Rajan needed 4 doses of medication each costing 2,250. Today he is able to smile because somebody was willing to help him.

12 years old Varun could not believe his own eyes. He was going to meet his favourite heroes. He was so excited about the whole event and shared it with his family & everybody who came to meet him. One couldn't miss the joy and the gleam in his eyes. The D - day arrived and he Along with 5 other children met Sachin Tendulkar, Wasim Akram, Mushtag Ahmed, and Saeed Anwar, before their time ran out. Can-Wish's aim is to fulfil such dreams ; whether its meeting their heroes, getting a new book or toy or just having an ice cream. YOU TOO COULD MAKE A DIFERENCE AND BRING A SMILE BACK ON A CHILD'S FACE.

Cancer causes pain, anguish, distress and agony. This is why we are there to help out to touch those affected with cancer and to fight cancer in all its guises.

Our Projects include :

Counselling : Trained volunteers are there to listen and console.

Awareness / Screening Camps : Can - Stop are organised and conducted by doctors and volunteers in the community.

Educational Therapy : invovles a play area for the children at ICH where educational material is provided to help children spend productive time during their hospital stay. Play materials include blocks, puzzles, books and drawing material.

Music Therapy : is conducted for the children once a month by Ramana Sunritya Aalaya, creative centre for Arts and Creative Movement Education.

Occupational Therapy : A basic craft is taught to the mothers' of the children at ICH to help them spend time productively.

Milky Way : A nutrition project provides milk with complan and biscuits for 60 children four times a week.

Tender Cocount Water is provided to 120 women once a week at Women's Hospital Egmore.

Blood Bank : It helps in obtaining blood for children and women when necessary.

Drug Bank : A corpus fund created to provide necessary drugs on a regular basis to all needy cancer affected children from low socio- economic background.

Can - Wish : fulfillment of wishes for the critically ill cancer children is provided between the age group of 0-16 years.

Sponsor A Child : this project helps children with good prognosis from a low Socio Economic - background. It provides essential drugs for a period of one year through individual sponsors.

Helpline : The public reach out to us through the help line. We provide counseling, second opinion, referal services, home visits and any other related information.

We want to make a difference in the lives of cancer affected patients by being there for them and together we can overcome pain.

We wish to inform you that any donations in cash/cheque towards "CAN-STOP" are eligible for 100% Tax Exemptions Under Section 35AC of Income Tax Act. Cheques should be drawn in favour of **SUNDARAM MEDICAL FOUNDATION - CAN - STOP.**

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CHICKENPOX can be prevented!

Why you and your family should be aware of Chickenpox (Varicella) and Shingles (Herpes Zoster)

What does varicella-zoster virus cause ?

The primary manifestation of varicella zoster virus is varicella, commonly known as chickenpox, which is a viral disease caused when an individual is first exposed to the virus. It presents as fever with a characteristic vesiculo-papular (blistered), highly pruritic (itchy) rash and is highly contagious. Infection of the lesions can result in permanent scarring.

The virus then remains latent in the body and can later be reactivated and cause herpes zoster (shingles).

What is the difference between varicella and herpes zoster ?

During a varicella infection (chickenpox), the sensory nerve cells become infected with viral particles, an association that persists throughout the individual's life. The viral particles then stay dormant in the sensory nerve cells. The virus can be reactivated later in life because the efficiency of the immune system declines with age. Hence herpes zoster (shingles) is more common in older people.

The main physical difference between the two infections is the rash and the pain associated with it. In shingles, the skin blisters form in red, inflamed groups along a nerve or group of nerves, and are extremely painful. In chickenpox, the rash is much more diffused, spreading over the body and the face.

How is varicella transmitted ?

The varicella virus is specially adapted to attack the mucous tissues of the upper respiratory tract. The virus spreads easily from person to person by airborne droplets, generally from the mucous secretions of the respiratory tract during coughing or sneezing. It also spreads by direct contact with varicella or herpes zoster lesions.

Who catches varicella ?

Everybody who has never had chickenpox before. This can be children, adolescents or adults.

What are the clinical signs of chickenpox ?

The characteristic symptom is an irritating itchy rash which starts on the trunk and gradually spreads over the face, where it can involve the scalp, mouth and ears, and also the upper arms and legs. Most children have 250-500 lesions, which form a crust after four to five days and remains for one to two weeks. Children may be quite distressed by the itching and can develop fever, chills, nausea and vomiting.

Does chickenpox cause complications in children ?

Although for many children varicella does not produce major health problems, complications can develop in some cases, especially pneumonia, which may be fatal. Bacterial superinfection of the skin, which may cause unsightly scarring, particularly of the face, may cause cosmetic concerns later in life, especially during adolescence.

What happens if an adolescent or adult contracts varicella ?

Varicella is more serious among adolescents and adults than in children. The fever is higher and continues for a longer period and the rash is much more severe. There is also a greater likelihood of complications such as pneumonia. But now, all of the above can be avoided.

Is it really necessary to prevent chickenpox?

There are three reasons. First, the full impact of childhood varicella on patients and on their families is often not appreciated. Physically, chickenpox is very uncomfortable for patients and may get complicated with secondary infections leading to scarring. The risk of serious complications is especially higher with increasing age.

Secondly, chickenpox can lead to loss of school days for children and loss of working days in adults. Epidemics tend to break out during late winter and early spring time, which often are the months for school/college examinations. Complications of the disease are associated with increase in cost of treatment and are an unnecessary financial burden.

Finally, when a simple solution is available, why take a chance? Now chickenpox can be prevented easily by vaccination. CONSULT YOUR DOCTOR ABOUT IT.

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